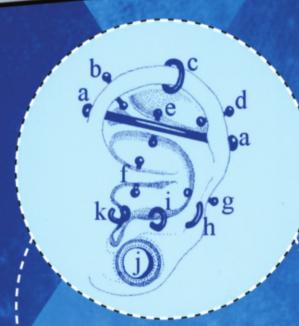
## guy n. rutty

essentials of autopsy practice



Recent advances, topics and developments



### **Essentials of Autopsy Practice**

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# **Essentials of Autopsy Practice**

**Recent Advances, Topics and Developments** 



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#### **Preface**

The public perception of a pathologist is largely based upon the ever-increasing number of television programmes depicting autopsy practice, be they fact or fiction, that are broadcast each day around the world. Thus, in the public's mind, a pathologist is a person that only performs autopsies, and yet this work is part of a subspecialism within the profession, and in the case of forensic pathology, upon which most television programmes are based, this accounts for a very small number of individuals undertaking very specialised investigations. The other general misconception concerning autopsy work is that it remains unchanged, i.e., the practice is largely as it has been for at least the last 100 years, with little advancement or development. Nothing can be further from the truth. In this second volume in the series on autopsy practice, subjects have again been chosen to assist all involved in post-mortem, crime investigation and bereavement work, specifically in areas where information may not be readily to hand, or in areas which may prove difficult for interpretation and where there have been advancements in practice over recent years. The book is again designed to be of use to trainee and consultant pathologist alike, be they a generalist or specialist, as well as to nurses, paramedical personnel, bereavement officers, lawyers and police. As this series evolves, each volume is intended for all involved with the dead and post-mortem work and hopes to assist with keeping one up to date with changing issues related to autopsy practice.

> G.N. Rutty Leicester, 2003

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## 1. Religious Attitudes to Death: What Every Pathologist Needs to Know. Part 2

J.E. Rutty

The most barbarous and the most fantastic rites and the strangest myths translate some human need, some aspect of life, either individual or social...In reality, then, there are no religions which are false. All are true in their own fashion; all answer, though in different ways, to the given conditions of human existence.

Emile Durkheim 1858-1917 [1]

#### Introduction

Special significance has always been given to the disposal of the dead throughout history and in every human society. The practice originates back to the ideas enthused by primitive people regarding human nature and destiny as opposed to sanitary concerns, when the disposal of the dead was ritualistic. Examples can be seen when studying, for example, the Palaeolithic people, such as the Neanderthals and later groups, because as well as burying their dead they also provided them with food, weapons and other implements. This implies that there was a belief that the dead still needed such things beyond the grave with such significant practices being traced back to approximately 50,000 BC. Practiced in most parts of the world, the ritual burial of the dead seems to stem from an instinctive incapacity or denial on the part of man to believe that death is the ultimate conclusion of human life. Additionally, the belief by man that something or some part of the individual person who has died continues to exist in some way perseveres today, even though there is clear scientific knowledge of physical decomposition caused by death itself. In contrast, the idea of personal extinction through death is a difficult concept that was unfamiliar until the sixth century BC, when it developed in the metaphysical considerations of Indian Buddhism. Such beliefs were not evident in the ancient Mediterranean world before its presentation by the Greek philosopher Epicurus (341-270 BC).

Profoundly influencing the thoughts, emotions and actions of mankind has been the idea that man can survive death. All religions, both past and present, hold such beliefs describing how man should behave and what his place is within the universe. These evaluations and descriptions are reflected in mortuary rituals and funerary customs which also take into account the practical processes to support the dead in reaching their destiny. Some beliefs will go even further and consider how such processes can help in saving the living from the dead who have been transformed into a different state of being; Shintoism being an example<sup>2</sup>.

In Britain today the majority of the world's religions continue to be represented and so it is not surprising that, in a diverse multi-cultural society such as our own, it remains extremely easy to cause offence unknowingly. It is vital that all histopathologists be aware of the key beliefs to religions common in our society so that the care of the deceased and relatives continue to remain respectful and spiritually sensitive. Understanding other people's religious beliefs can help us to interpret their beliefs and behaviours contextually. Unfamiliarity with other religions can cause misunderstanding, missed cues and bring communication to an end, leaving the histopathologist as a stranger and an outsider instead of someone the family can trust<sup>2</sup>.

The aim of this second chapter is again not to enter into a theological debate, but to provide a descriptive account that can be easily accessed and used for reference purposes regarding spiritual care of the deceased in Britain today. My previous chapter presented the three broad categories of contemporary faiths being practiced, these are the Abrahamic, Vedic and other major traditions<sup>2</sup>. This resulted in ten of the major religions and/or cultures being described previously which encompassed Judaism, Christianity and Islam; Hinduism, Buddhism and Jainism; Sikhism, Shintoism, Taoism, and the Baha'i Community, respectively.

To remain accessible for everyday use by histopathologists, this chapter concentrates on those religions and/or cultures that are less common in the UK, but of course are of no less importance. In alphabetical order, some of those religions will be presented concerning the key beliefs and the meaning of death, care of the body, funerals, organ donation and autopsies. This chapter does not claim to be in-depth and to provide all the answers. Instead, it is recommended that this information is used in conjunction with the views of the relatives and/or friends of the deceased.

#### **Afro-Caribbean Community**

The main religion of the Caribbean islands is some form of Christianity, but different island communities have differing religious backgrounds and wide variations in their ritual practice. The major Christian churches in this community are Anglican, Methodist and Pentecostal. Overall, these communities are likely to be more expressive in their practice of religion than their Caucasian counterparts. The extended family influence can be quite complex, but strong, good relations are maintained with the family coming together when one of its members dies<sup>3</sup>.

#### **Death**

In the UK, the religious background of an Afro-Caribbean community will reflect the culture of the island from which its members came. At death, religious differences are likely to be minimised, and cultural and island identity predominate. Close relatives may wish to be present at the time of death, so one must be aware that there may be more than the average number of visitors for the dying or deceased.

#### **Care of the Body**

Routine last offices are appropriate. There are no religious objections to the body being handled as long as respect is shown. It may be preferred that a nurse from a similar ethnic background is able to fulfil this duty. The body should ideally be embalmed, especially if the funeral will be delayed.

#### **Funerals**

In the UK, burial is the preferred method of disposal. The funeral is an important and elaborate occasion for the extended family and for all those who loved the deceased during life. It may be that the entire community would wish to attend. The body may be viewed at home before the funeral service and again during the church ceremony characterised by music and tributes. At the graveside the family will fill in the grave themselves while singing continues<sup>3</sup>.

#### **Organ Donation**

Those with belief in the sanctity of the body are unlikely to agree to organ donation. Younger family members may have different views.

#### **Coroner's Enquiry and Autopsies**

Older members of the community may believe that the body must be intact for the after-life and will be deeply offended by its disfigurement. They are unlikely to give consent for post-mortem except for coroner's cases. Again, different views may be held by younger family members.

#### **Atheism**

Most histories of atheism choose the Greek and Roman philosophers Epicurus, Demoncritus, and Lucretius as the first atheist writers. There are many types of atheism, including: humanism, secularism, rationalism, Buddhism, humanistic Judaism, Christian non-realism, postmodernism and unitarian universalism. Atheism is not a religion, nor a specific philosophical system. Atheism is the absence of a belief in God. Followers of this belief do not believe in God or other spiritual beings and do not use God to explain the existence of the universe. Some atheists go further and deny that God, or other spiritual beings, exist.

Atheists hold that there is no evidence for the existence of any God or gods; instead there are many good arguments that prove the contrary resulting in the topic being meaningless. In 1841 Ludwig Feuerbach argued that God was a human invention, a spiritual device to help us deal with our fears and aspirations. It was the theory of evolution put forward by Charles Darwin (1859) and other scientific discoveries that undermined the value of religion as a way of explaining the nature and existence of the world. Anthropologists, too, were casting doubt on previous certainties. Research into comparative religion revealed that there was a great deal of similarity between the rituals and stories of many religions – even tribal religions seemed to have elements in common with Christianity. This posed the big problem of how could any religion claim that is was the only true faith, and how could any

religion claim to be the unique result of God's revelation, since all religions seemed to share so much in common.

Atheists believe that human beings can devise suitable moral codes to live by without the aid of gods or scriptures. It is perfectly possible to be both religious and an atheist. Virtually all Buddhists manage it, as do many members of other faiths. But many atheists are also secularists, and are hostile to any special treatment given to organised religion. Atheists, though, do exhibit strong family values, with research showing that they have one of the lowest divorce rates compared to any religious group<sup>4</sup>.

#### Death

Atheists believe that more traditional religious beliefs are often supported by fear of external punishment after death. Such fear of retaliation by an angry and vengeful god during this lifetime means to atheists that people live their lives in fear, which is unhealthy.

#### Care of the Body

Routine last offices are appropriate. There are no rites at death.

#### **Funerals**

Many atheists will feel uncomfortable with religious funerals if religion had no meaning for the dead person, particularly if these views are also held by the person's relatives and friends. Instead, a humanist funeral will remember the life of the person who has died and will reflect on their contribution to the world and others. No hymns or prayers will be included. Instead, it may include: music; a non-religious reflection on death; readings of poetry and prose; reminiscences; a eulogy; ritual actions such as candle lighting; formal words of goodbye. Very importantly, it provides a time for family and friends to share their sadness and to create a bond of support for those who were closest to the dead person. It is the individual's decision to choose burial or cremation<sup>5</sup>.

#### **Organ Donation**

No objection, but it is the individual's decision.

#### **Coroner's Enquiry and Autopsies**

No objection, but it is the individual's decision.

#### **Church of Jesus Christ of Latter-day Saints**

After arriving in Britain at the beginning of the reign of Queen Victoria, the Church of Jesus Christ of Latter-day Saints has over 11 million members worldwide, including over 180,000 in the UK. It is believed to be one of the fastest growing religions in the world with a prediction by 2080 of a membership of 265 million people. Their headquarters are in Salt Lake City, Utah, USA.

The Church of Jesus Christ of Latter-day Saints was founded in 1830 in New York State (USA) by Joseph Smith who believed that he received a revelation from God through an angel (Moroni) and a book inscribed on golden plates. These gold plates gave an account of the revelations of many prophets largely completed by the prophet historian Mormon. Subsequently, Mormon's son, Moroni, added to the plates before concealing them in a hill where they lay for fourteen centuries. In 1823 the resurrected being of Moroni appeared before Joseph Smith instructing him about the plates. Smith translated these inscriptions into English, returned the plates to Moroni and published the Book of Mormon telling the story of the ancient people of America. Along with the Book of Mormon, the Old and New Testaments are the central beliefs of the Church. The Church today is centred on Christ, but has very large differences in beliefs when compared to the Catholic, Protestant and Orthodox Christian Churches. Mormons believe that the Church of Jesus Christ of Latter-day Saints restores the Church as conceived by Christ, whereas other Christian churches have gone astray.

Mormons do not believe that the life of a human being begins with their earthly birth. Instead, they believe that we have an eternal life stretching either side of our lives on earth resulting in three stages of life: pre-existence as a spirit child, a time of probation on earth and eternal life with the Heavenly Father. This governs their whole way of life<sup>3</sup>.

#### Death

There is a belief that at some time after death, the spirit and the body will reunite and be resurrected to the spirit world. The spirit world is divided in two: Paradise, where the good spirits wait for resurrection; and Spirit Prison, where the unrighteous spirits live in darkness. The good spirits are able to visit them there and preach the gospel. If one of the unrighteous spirits accepts the gospel and repents they are able to move to the other part of the world.

Mormons consider that living on earth is a time to prove oneself as being worthy in order to eventually return to live in the presence of Jesus Christ and God the Father. Family unity is of immense importance, signified by the "sealing" ceremony at the Temple. This is when man and wife are sealed together for eternity, along with their children and other family members who may already be dead, in order that they may all be together when resurrected. Death therefore is seen as only a temporary separation from their loved ones. Death is considered also to be a blessing and a purposeful part to eternal existence. There is no ritual for the dying, but spiritual contact is important<sup>3</sup>.

#### Care of the Body

There are no special rituals associated with dying or death for any age group, including the need for emergency baptism. Routine last offices are appropriate, however, the sacred garment, if worn, must be replaced on the body afterwards.

#### **Funerals**

It is believed to be proper to bury the dead in the ground. Cremation is discouraged, but not forbidden. The Bishop will offer comfort and practical assistance with funeral arrangements. The body may be viewed before the funeral, with family prayers before the service, which usually takes place at the church meeting house.

#### **Organ Donation, Coroner's Enquiry and Autopsies**

Individuals are encouraged to evaluate the pros and cons, to implore the Lord for inspiration and guidance, and then to take the course of action that would give them a feeling of peace and comfort.

#### Note

Individual members of the church do not mind being called Mormons, but they prefer their church to be referred to as The Church of Jesus Christ of Latter-day Saints or the Church of Christ, not the Mormon Church.

#### Confucianism

Developed from the teachings of Confucius and his disciples, Confucianism is a major system of thought in China. Confucianism is concerned with the principles of good conduct, practical wisdom and proper social relationships. Confucianism has influenced the Chinese attitude toward life, set the patterns of living and standards of social value, and provided the background for Chinese political theories and institutions. It has spread from China to Korea, Japan, and Vietnam and has aroused interest among Western scholars. There are about six million Confucians in the world today<sup>6</sup>.

Although Confucianism became the official ideology of the Chinese state, it has never existed as an established religion with a church and priesthood. Chinese scholars honoured Confucius as a great teacher and sage but did not worship him as a personal god. Nor did Confucius himself ever claim divinity. Unlike Christian churches, the temples built to Confucius were not places in which organised community groups gathered to worship, but public buildings designed for annual ceremonies, especially on the philosopher's birthday.

The principles of Confucianism are contained in the nine ancient Chinese works handed down by Confucius and his followers, who lived in an age of great philosophic activity. These writings can be divided into two groups: The Five Classics and the Four Books. The keynote of Confucian ethics is *jen*, variously translated as "love", "goodness", "humanity", and "human-heartedness". *Jen* is a supreme virtue representing human qualities at their best. In human relations, construed as those between one person and another, *jen* is manifested in *chung*, or faithfulness to oneself and others, and *shu*, or altruism, best expressed in the Confucian golden rule,

Do not do to others what you do not want done to yourself 6.

After the death of Confucius in the early fifth century BC, various schools of Confucian thought have emerged through the centuries including Mencius, Hsuntzu, Han Confucianism and Neo-Confucianism.

#### Death

Beliefs about death will vary depending upon the school of Confucian thought followed.

#### Care of the Body

At death, relatives cry out aloud to inform the neighbours. The family starts mourning and puts on clothes made of a coarse material. The corpse is then washed and put in a coffin. Food and significant objects of the deceased are placed into the coffin.

#### **Funerals**

Mourners will bring incense and money to offset the cost of the funeral. A Buddhist or Taoist priest (or even a Christian minister) performs the burial ritual. Friends and family follow the coffin to the cemetery, along with a willow branch which symbolises the soul of the person who has died. The latter is carried back to the family altar where it is used to "install" the spirit of the deceased. Liturgies are performed on the seventh, ninth, and 49th day after the burial and on the first and third anniversaries of the death<sup>7</sup>.

#### **Organ Donation**

No objection, but traditionalists may be superstitious.

#### **Coroner's Enquiry and Autopsies**

No objection, but traditionalists may be superstitious.

#### Jehovah Witnesses

Many Jehovah's Witnesses beliefs are similar to those of Fundamentalist Christianity. They try to live their lives according to the Old and New Testament and are deeply religious. They believe that Jehovah is the Supreme Being and that Jesus is the Son of God who was a created being, but to have originally existed in a pre-human state as the Archangel Michael. The Holy Ghost is not a separate entity, but is simply the way in which God interacts with the world.

It is held that the Heavenly Kingdom took effect in 1914 with the invisible enthronement of Christ as King. It is currently occupied by an Anointed Class of about 135,400 people, the selection of which was completed in 1935, leaving to date 8,600 still living on Earth.

Jehovah Witnesses reject the symbol of Christianity, the cross, as being of pagan origin, believing instead that Jesus was crucified on a single upright wooden stake. Christ's second coming was not a physical return to Earth, but an invisible event in 1914 when Satan and Christ engaged in a heavenly battle. Afterwards, Christ began the Heavenly Kingdom and Satan was expelled to Earth, with World War I being a visible sign of Satan's earthly wrongdoings.

Jehovah Witnesses believe that in the near future, the battle of Armageddon will begin, where Jesus under Jehovah's divine rage will execute vengeance upon followers of all other, i.e., false, religions. Following this great suffering, it is believed that the world will be purified and God's Kingdom will be established on earth for 1,000 years. Currently, they regard the world as being under the control of Satan and so will not run for public offices, vote in elections or join the Armed Forces.

Jehovah Witnesses recognise only one day of celebration that is the Memorial of Christ's Death at the time of Passover. Members found celebrating "Pagan" holidays such as Christmas, Halloween, etc. may be excommunicated. Not having a Sabbath, Jehovah Witnesses regard all days as holy having five meetings every week: public talk, watchtower study, theocratic ministry school, service meeting and book study.

#### Death

People who die before Armageddon cease to exist. Jehovah Witnesses believe that at death their body deteriorates and returns to dust. However, at the time of resurrection, God will create a new body for each believer, similar to their former shell, but without the imperfections, resuming life with their original personality and memories<sup>3</sup>.

#### **Care of the Body**

No official last rites are practiced when death occurs. Routine last offices are appropriate.

#### **Funerals**

Both burial and cremation are acceptable. There is no formal written service, instead each is prepared on an individual basis.

#### **Organ Donation, Coroner's Enquiry and Autopsies**

No definite statement related to this issue. Neither is encouraged, as it is believed to be a matter for the individual conscience, but it is unlikely. All organs and tissues, though, must be completely drained of blood before transplantation.

#### Note

- Many conservative faith groups do not give equal opportunity for women to lead and so the authority in the family tends to be reserved for men only.
- Blood represents life itself and must be handled with respect. It is not acceptable for it to be stored or reused<sup>3</sup>.

#### **New Age Spirituality**

Unlike most formal religions, the New Age Movement has no holy text, central organisation, membership, formal clergy, creed, etc. Instead, the New Age is a free-flowing spiritual movement. It is a network of believers who share similar beliefs and practices which they tend to add on to whatever other formal religion they follow. Some of the beliefs include astrology as a method of foretelling the future, crystals as a source of healing and Tarot cards as a base for life decisions. They believe, too, that God is a state of higher consciousness and that a person may reach or can grow to the total realisation of personal, human potential.

New Age Spirituality began in England in the 1960s. Its aim is to provide spiritual and ethical guidance for the future due to the belief that other religions had failed them. There are a number of fundamental beliefs, but individuals are encouraged to choose which ones suit them best, e.g., monism (all that exists comes from a single divine energy), pantheism (all that exists is God), panentheism (God is all that exists), reincarnation, karma (the good and bad deeds that we do is accumulated in our record), aura, personal transformation (mystical experience), crystals, meditating, new-age music (harp, flute) and holistic health<sup>9</sup>.

#### Death

Beliefs about death are dependent on what formal underlying religion New Age Spiritualists follow.

#### **Care of the Body**

This is dependent on what formal underlying religion New Age Spiritualists follow.

#### **Funerals**

This is dependent on what formal underlying religion New Age Spiritualists follow.

#### **Organ Donation**

This is dependent on what formal underlying religion New Age Spiritualists follow.

#### **Coroner's Enquiry and Autopsies**

This is dependent on what formal underlying religion New Age Spiritualists follow.

#### **Raelians**

On the 13th December 1973, French journalist Claude Vorilhon (named Rael by an extra-terrestrial) held that he was contacted by a visitor from another planet who asked him to establish an Embassy to welcome these people back to Earth. The extra-terrestrial told Rael that they were the ones who made all life on Earth and were the origins of many religions. Now that humans had matured they wished to make official contact through an embassy.

The message in essence explains how life on Earth was not a random result of evolution or work of a supernatural God, but instead a deliberate creation using DNA for genetic manipulation and cloning to make human beings literally in their own image, i.e., scientific creationism. It is held that their work can be found in many cultures, e.g., in the Book of Genesis and the biblical account of creation, the word "elohim" translated as God in the singular, but as "those who came from the sky" in the pleural.

It is attested that these extra-terrestrials then left Earth to enable humanity to develop alone. However, they maintained contact through prophets including Buddha, Moses, Jesus and Mohammed with the intention of progressively educating humanity. Now that man has been to the moon and also created life through

DNA cloning, the extra-terrestrials believe that the human race is capable of understanding their alien creators rationally rather than mystifying them or adoring them. It is held that the Elohim created us to be happy, to blossom and in turn to become creators. Raelians believe that today human beings are immersed in permanent suffering caused by the rules of our society. Instead, we should dedicate ourselves to think, to create, to blossom, to love and to seek pleasure with the utmost respect for others<sup>10</sup>.

#### Death

This is dependent on individual beliefs. However, there is a fundamental belief that there is a possibility of eternal life due to the scientific development of human cloning<sup>11</sup>.

#### Care of the Body

This is dependent on individual beliefs.

#### **Funerals**

This is dependent on individual beliefs.

#### **Organ Donation**

This is dependent on individual beliefs. However, it may be considered a virtuous act as it may promote or save somebody else's life.

#### **Coroner's Enquiry and Autopsies**

This is dependent on individual beliefs. However, it may be considered a virtuous act as it may promote or save somebody else's life.

#### Rastafarianism

Rastafarianism is most often associated with dreadlocks, smoking of marijuana and reggae music. However, it is much more than simply a religion of Jamaica. It began in the Jamaican slums and has since spread throughout the world. Rastafarianism currently has a membership of over 700,000 worldwide. Rastafarianism began in the 1930s in the West Indies, among the descendents of slave families who had come from Africa. Identification with Africa is central to the Rastafarian doctrine and the movement is linked to the roots of resistance to slavery<sup>3</sup>.

In the early 1920s, Marcus Garvey (1887–1940), an influential black spokesman and founder of the "Back-to-Africa" movement, often spoke of the redemption of his people as coming from a future black African king. Only a few years later that prediction was fulfilled in the accession of Ras (Prince) Tafari (1982–1975) as the Emperor of Ethiopia (Haile Selassie I) on November 2nd in 1930. He is considered to be a divine being, the Messiah of the human race, who will ultimately lead all black people to freedom. Believers claim there is a direct lineage from the biblical King David to Ras Tafari (hence the name Rastafarian).

The Rastafarian movement has rejected many aspects of the major cultural influences which were predominant in Jamaica and has become a distinct entity. The Old and New Testaments are still regarded as scriptures, but they do not consider themselves to be Christians. For them, Christ's spirit has been reborn in Ras Tafari, the new Messiah<sup>3</sup>.

One of the key doctrines of Rastafarians is the belief that they are the true Jews, who will eventually be redeemed by repatriation to Africa, their true home and Heaven on Earth. However, an important historical event occurred when Haile Selassie visited Jamaica on 21st April 1966. He convinced Rastafarians not to seek to immigrate to Ethiopia until they had liberated the people of Jamaica. Since then the 21st April has been celebrated as a "special holy day" among Rastafarians.

The belief system of Ras Tafari was vague and loosely defined, even at its inception, due to its lack of a single authoritative voice, that what was to be acceptable doctrine was largely a matter of individual interpretation. Early in the history of the movement, Leonard Howell gave the Rastafarians six principles: Hatred for the whites; Complete superiority of the Black race; Revenge on Whites for their wickedness; The negation, persecution, and humiliation of the government and legal bodies of Jamaica; Preparation to go back to Africa; Acknowledging Emperor Haile Selassie as the Supreme Being and only ruler of Black people.

Rastafarianism is a personal religion. There are no churches, set services or official clergy. All members share in the religious aspects, have a deep love of God and believe that the Temple is within each individual<sup>12</sup>.

#### Death

Visiting the sick often in groups is important. The family may pray by the bedside of a dying member, but other than this there are no last rites. Rastafarians believe in the resurrection of the soul after death, but not of the flesh.

#### Care of the Body

Routine last offices are appropriate.

#### **Funerals**

Burial is preferred, but cremation is not forbidden. The funeral is plain and simple and attended only be intimate family and friends.

#### **Organ Donation, Coroner's Enquiry and Autopsies**

Both organ donation and autopsy are considered distasteful. Few would agree to either, except when ordered by the Coroner. Members of the faith are readily identified by their distinctive hairstyles. "Dreadlocks" or "locks" are a symbol of faith and a sign of black pride. Orthodox members may not permit their hair to be cut<sup>3</sup>.

#### Note

• There is a taboo on the wearing of second-hand clothing and orthodox patients may even be unwilling to wear hospital garments which have been worn by others. Disposable gowns may be preferred.

• Marijuana is considered to be the "holy herb" and its smoking is a holy sacrament to many. The herb is believed to be the key to new understanding of the self, the universe and God. It is the vehicle to cosmic consciousness<sup>3</sup>.

#### Vodun

Vodun is commonly called Voodoo by the public. The name is traceable to an African word for "spirit". Voduns can be traced directly to the West African Yoruba people more than 6,000 years ago. Today there are over 60 million people who practice Vodun worldwide. Vodun, like Christianity, is a religion of many traditions. Each group follows a different spiritual path and worships a slightly different pantheon of spirits, called Loa. Loa means "mystery" in the Yoruba language.

Yoruba's traditional belief includes a chief God Olorun, who is remote and unknowable and who authorised a lesser God Obatala to create the Earth and all life forms. There are hundreds of minor spirits. Followers of Vodun believe that each person has a soul which is composed of two parts: a "gros bon ange" or "big guardian angel" and a "ti bon ange" or "little guardian angel". The latter leaves the body during sleep and when the person is possessed by a Loa during a ritual. There is concern that the "ti bon ange" can be damaged or captured by evil sorcery while it is free of the body.

The purpose of rituals is to make contact with a spirit, to gain their favour by offering them animal sacrifices and gifts, and to obtain help in the form of more abundant food, higher standards of living, and improved health. Human and Loa depend upon each other; humans provide food and other materials; the Loa provide health, protection from evil spirits and good fortune. Rituals are held to celebrate lucky events, to attempt to escape a run of bad fortune, to celebrate a seasonal day of celebration associated with Loa, for healing, at birth, marriage and death.

Vodun priests can be male ("houngan") or female ("mambo"). A Vodun temple at its centre has a pole ("Poteau-mitan") where the God and spirits communicate with the people. An altar will be elaborately decorated with candles, pictures of Christian saints, and symbolic items related to the Loa. Rituals consist of some of the following components:

- A feast before the ceremony; creation of a veve.
- A pattern of flour on the floor which is unique to the Loa for whom the ritual is to be conducted.
- Shaking a rattle and beating drums which have been cleansed and purified.
- Chanting; dancing by the priests which typically builds in intensity until one of them becomes possessed by Loa and falls. This is when their "ti bon ange" has left their body and the spirit has taken control. The possessed dancer will behave as the Loa and is treated with respect and ceremony by the others present.
- Animal sacrifice of either a goat, sheep, chicken or dog. They are usually humanely killed by slitting their throat, when the blood is collected in a vessel. The possessed dancer may drink some of the blood so that the hunger of the Loa is believed to be satisfied. The animal is then usually cooked and eaten. Animal sacrifice is a method of consecrating food for consumption by followers of Vodun, their gods and ancestors.

The Vodun priests confine their activities to "white" magic, which is used to bring good fortune and healing. However, caplatas perform acts of evil sorcery or black magic, sometimes called "Left-handed Vodun". Rarely, a priest will engage in such sorcery, but a few will alternate between white and dark magic<sup>13</sup>.

#### Death

One belief unique to Vodun is that a dead person can be revived after having been buried. After resurrection, the zombie has no will of their own, but remains under the control of others. In reality, a zombie is a living person who has never died, but is under the influence of powerful drugs administered by an evil sorcerer. Although most Voduns believe in zombies, few have ever seen one. There are a few recorded instances of persons who have claimed to be zombies. Sticking pins in "voodoo dolls" was once a method of cursing an individual by some followers of Vodun and occasionally the practice continues today<sup>13</sup>.

#### **Care of the Body**

This is dependent on individual beliefs which will vary according to the deceased roots, background and family upbringing.

#### **Funerals**

Usually burial is preferred.

#### **Organ Donation**

This is dependent on individual beliefs which will vary according to the deceased roots, background and family upbringing.

#### **Coroner's Enquiry and Autopsies**

This is dependent on individual beliefs which will vary according to the deceased roots, background and family upbringing.

#### Zoroastrianism

Founded by Zoroaster or Zarathustra and originating in the sixth century BC, Zorastrianism was the dominant religion of Persia until the rise of Islam. Today, the largest communities are in India (92,000) and Iran (around 30,000) and it is estimated that there are about 7,000 Zoroastrians in Britain, about 5,000 of whom live in London. Its scriptures are known as the "Avesta". It maintains a dualistic doctrine, contrasting the force of light and good in the world with that of darkness and evil<sup>14</sup>.

Zoroastrian practices include worship through prayers and symbolic ceremonies. Rituals are conducted before a sacred fire, as they regard the fire as a symbol of their God. Zoroastrians do not generally accept converts, as one has to be born into religion, although some members will dispute this. Members are dedicated to a threefold path, as seen in their motto "Good thoughts, good words, good deed". Offshoots of Zorastrianism include Mithraism and Manichaeanism<sup>15</sup>.

#### Death

After death, the soul is allowed three days to meditate on their life. The soul is then judged. If the good thoughts, words and deeds outweigh the bad, then the soul is taken into heaven, otherwise the soul is led to hell. There are no last rites before death, but Zoroastrians would wish to have their loved ones near at the time of death. Relatives, friends, or, rarely, a Zoroastrian priest may say prayers.

#### Care of the Body

Routine last offices are appropriate, however, it is important that the body is bathed before being dressed in white clothing. Most families will provide a special "sadra" which is to be worn next to the skin under the shroud, with the sacred "kusti". The family may wish the head to be covered with a cap or scarf<sup>3</sup>.

#### **Funerals**

Funerals should take place as quickly as possible after death, either on the same day or on the next. In the UK both burial and cremation are accepted. However, Zoroastrians believe that earth burial, cremation or disposal by water contaminates the sacred elements of the earth, fire and water.

In India, the Tower of Silence is the setting for disposal. This stone tower, outside the city, is built with three concentric circles, one each for men, women and children. It has no windows and no roof, and only certain bearers are allowed to enter. After ritual observances, the body is carried to the tower and left on the stone floor, exposed to the sun and the vultures who strip the bones clean within a couple of hours. The sun-dried bones are later swept into the central well<sup>3</sup>.

#### **Organ Donation**

Orthodox Zoroastrians consider that pollution of the body is against the will of God. They will be against blood transfusion for this reason, probably being unwilling both to donate and receive. Organ transplantation likewise introduces the concept of bodily or genetic pollution and is forbidden in strict religious law.

#### **Coroner's Enquiry and Autopsies**

Post-mortems are forbidden in religious law and would be refused except for coroner's cases. Body donation is likewise forbidden.

#### **Conclusion**

Within our own culture, each of us finds differing meanings in our life and ultimately our death. It is unhelpful and almost harmful to assume anything about what life events and death means for another person. However, understanding other people's religions can help us to interpret their beliefs and behaviours contextually. Unfamiliarity with another religion or culture can cause misunderstanding, missed cues and can bring communication to an end, leaving the histopathologist as a stranger and an outsider instead of someone the family can trust.

Health professionals can use their background knowledge and experience of a culture and/or religion to stimulate questions with relatives and/or friends of the deceased, but they must be careful to avoid stereotyping. Berling and Fowkes in 1983 remain to describe classical guidelines for use in clinical practice based on the acronym LEARN (listen, acknowledge, recommend and negotiate)<sup>16</sup>.

In conclusion, the final decision about autopsy always rests with the family unless an autopsy is required under the legal authority of Her Majesty's Coroner. Histopathologists need to remain alert for religious influences on the family at the time of a patient's death, so that care can remain spiritually sensitive. Such sensitivity will win the appreciation of families and may increase autopsy rates<sup>17</sup>.

Table 1.1 Summary of religions.

Religion	Care of the body	Funerals	Organ donation	Autopsy
Afro-Caribbean Community	Routine last offices preferably by a nurse from a similar ethnic background. Body ideally embalmed	Burial preferred. Body maybe viewed at home and/ or in Church	Unlikely to agree.	Younger members may have different views
Atheism	Routine last offices are appropriate	Burial or cremation. Humanist funeral	No objection	
Church of Jesus Christ of Latter-day Saints	Routine last offices with a sacred garment being worn, replaced on the body afterwards	Burial is preferred with cremation being discouraged, but not forbidden	Individual decision	
Confucianism	May prefer to be washed by the family	Burial is preferred	No objection, but traditionalists may be superstitious	
Jehovah Witnesses	Routine last offices are appropriate	Burial or cremation	Up to individual conscience, but blood not acceptable in being stored or reused	
New Age	Dependent on underlying religion followed			
Spirituality Raelians	Dependent on individual beliefs			
Rastafarianism	Routine last offices are appropriate	Burial preferred, but cremation not forbidden	Organ donation and autopsy considered distasteful and so few would agree. Orthodox members may not permit their hair to be cut	
Vodun	Dependent on individual beliefs which will vary according to roots, background and family upbringing	Burial is preferred	Dependent on individual beliefs which will vary according to roots, background and family upbringing	
Zoroastrianism	Routine last offices are appropriate before being dressed in white clothing	Burial or cremation is acceptable	Probably unwilling to donate	Forbidden in religious law

#### References

- 1. Durkheim E. The elementary forms of religious life, trans. Joseph Ward Swain, cited in Ayer AJ, O'Grady J, editors. A dictionary of philosophical quotations. Oxford: Blackwell, 1994;121.
- 2. Rutty JE. Religious attitudes to death: What every pathologists needs to know. In: Rutty GN, editor. Essentials of autopsy practice. Part 1. London: Springer, 2001;1–22.
- 3. Green J. Death with dignity, vol. II: Meeting the spiritual needs of patients in a multi-cultural society. London: EMAP Healthcare Ltd, 1993.
- 4. Robinson BA (2001) Atheism, agnosticism, free thinking, humanism, etc. <a href="http://www.religioustolerance.org">http://www.religioustolerance.org</a>. (cited May 2002).
- 5. Wilson J. Funerals without God: a practical guide to non-religious funerals. Prometheus Books, 1991.
- 6. Yao X. An introduction to Confucianism. Cambridge University Press, 2000.
- 7. Jao X. Confucianism and Christianity. A comparative study of Jen and Agape. Sussex Academic Press, 1996.
- 8. Reed DA. Jehovah's Witness Literature: a critical guide to Watchtower Publications. Baker Book House, 1993.
- 9. Robinson BA. New Age Spirituality. http://www.religioustolerance.org (cited Dec. 2002).
- Raelian Movement. The message given by extra-terrestrials. Raelian Movement Publications, 1998.
- 11. Raelian Movement. Yes to human cloning: Eternal life thanks to science. Raelian Movement Publications, 2001.
- 12. Barret L. Rastafarians. Beacon Press, 1997.
- 13. Turlington C. The complete idiots guide to Voodoo. Alpha Books, 2001.
- 14. Varza B. Zoroastrian faith and philosophy. <a href="http://www.religion-info.net">http://www.religion-info.net</a>. (cited December 2002).
- Boyce M. Zorastrians: Their religious beliefs and practices. Routledge, Taylor and Francis Books, 2001.
- Berling EA, Fawkes WC. A teaching framework for cross-cultural health care. West J Med 1983;139:934–8.
- 17. Perkins HS. Cultural differences and ethical issues in the problem of autopsy requests. Texas Med/The Journal 1991;87:72–7.

## 2. The Investigation of Inherited Metabolic Disease After Death

S.E. Olpin, M.-J. Evans

#### Introduction

The term "inborn errors of metabolism" was first used by Garrod in 1909 to define a spectrum of genetically inherited disorders characterised by blocks in the metabolic pathway due to deficient activity of an enzyme in each pathway¹. Initially, he described just four disorders – alkaptonuria, albinism, cystinuria and pentosuria. As our knowledge of the human genome has expanded, there has been a vast increase in the number of diseases defined and which may now be diagnosed. It is beyond the scope of this chapter to deal with each of these disorders individually; instead, we hope to raise awareness of the possibility of an underlying metabolic disease and to provide a framework for appropriate investigation.

Many of these disorders are inherited as autosomal recessive or x-linked traits. A few are autosomal dominant. Mitochondrial disorders form a genetically distinct group as their enzymes are encoded both by the nuclear genome and by the maternal mitochondrial DNA.

Inherited metabolic disorders have profound effects not only on patients and their immediate family, but also on the long-term reproductive health of that family, often with implications for the extended family. Only by accurate diagnosis can appropriate genetic counselling and antenatal diagnosis be made available to these individuals.

A coordinated plan of investigation must begin with accurate clinical and family histories. This must include information on ethnicity, consanguinity and previous obstetric history, physical examination and preliminary post-mortem findings together with the results of any pre-mortem biochemical and haematological testing. This information dictates the direction of subsequent investigations. Strategy for diagnosis consists of using all available information to direct the selective use of appropriate laboratory testing. It is our intention to provide a framework for assessing possible underlying defects, particularly those that are more likely to present as possible inherited metabolic defects to the histopathologist. We will therefore concentrate on:

- 1. Description of the more common inborn errors of metabolism.
- 2. Steps to diagnosis: Post-mortem findings suggestive of metabolic disease.
- 3. Post-mortem specimens and results.
- 4. Neonatal disease.
- 5. Disorders associated with Sudden Infant Death Syndrome (SIDS), (crib death Sudden Unexpected Death in Infancy (SUDI)).
- 6. Non-accidental injury and metabolic disease.
- 7. Cardiomyopathy and associated metabolic disorders.

It must be remembered that the investigation of suspected metabolic disease at post-mortem requires a coordinated plan of action. We cannot overemphasise the importance of good clinical liaison between the different disciplines in facilitating appropriate investigations and conserving precious post-mortem samples.

From a practical point of view the availability of electrospray tandem mass spectrometry has revolutionised the investigation of metabolic disease and is *the* major advance in the post-mortem investigation of these disorders, facilitating the identification of a wide range of metabolic diseases in tiny samples of blood, plasma and bile<sup>2</sup>.

#### **Common Metabolic Disorders – Summary**

Below, we aim to characterise the principal disorders encountered in clinical practice.

#### **Fatty Acid Oxidation Disorders**

The major metabolic flux of long-chain fatty acids is through the β-oxidation system of the mitochondria present in all cells except the mature erythrocyte. Longchain fatty acids are an important source of respiratory fuel for many tissues, especially skeletal and cardiac muscle. Ketone bodies act as a vital energy source in fasted individuals, particularly for the brain, which is unable to use fatty acids as fuel. Long-chain fatty acids require a carnitine-dependent transport system to cross the mitochondrial membrane into the matrix where the process of β-oxidation takes place. This group of disorders is an important cause of acute metabolic decompensation and sudden death in the neonatal period, infancy and early childhood<sup>3</sup>. Patients may present acutely with a life-threatening illness during intercurrent infections, operations or other catabolic stress, having apparently been previously well. The presentation may be one of sudden death with little or no illness. Hypoketotic hypoglycaemic coma leading to death occurs during such catabolic states. There may be signs of liver failure with hyperammonaemia and a Reyelike presentation. Oxidation defects, particularly of long-chain fatty acids, may affect skeletal muscle, causing chronic weakness as well as acute or chronic cardiomyopathy that can be either hypertrophic or dilated. Cardiac arrhythmias resulting in sudden death may be induced by accumulation of long-chain acylcarnitines, which are particularly arrythmogenic.

#### Medium-chain Acyl-CoA Dehydrogenase Deficiency (MCAD)

MCAD is the commonest fatty acid oxidation defect occurring in central Europe with an incidence as high as 1:8,000 live births. The first crisis is fatal in up to 25%

of cases, patients classically presenting with a Reye-like illness<sup>4</sup>. Some patients presenting initially as SIDS have subsequently on biochemical testing been shown to have MCAD<sup>5</sup>. Contrary to some older literature, babies can present within the first 48 hours of life as a sudden death, at least five such cases are known to us. Presentation after the age of five years is rare although there are isolated reports of adult presentations following extreme metabolic stress<sup>6,7</sup>. The diagnosis can be made by measuring cis-4-decenoic acid in plasma, analysis of urine organic acids and/or blood acylcarnitines followed by DNA confirmation. The common K304E mutation accounts for 85% of this disease in most of Western Europe<sup>8</sup>.

#### **Carnitine Deficiency and Other Fatty Acid Oxidation Defects**

There are approximately 15 other reported defects affecting the carnitine shuttle and oxidation of fatty acids<sup>9,10</sup>. Cumulatively these other defects are probably as common as MCAD but some ethnic groups will show a higher incidence of certain disorders, e.g., MCAD in Western Europeans, carnitine palmitoyl transferase deficiency type I (CPT I) in Asians. Cardiac involvement either as cardiomyopathy and/or arrhythmias (premature ventricular complexes, atrioventricular block, and ventricular tachycardia) is particularly associated with long-chain defects, e.g., long-chain 3-hydroxyacyl-CoA dehydrogenase deficiency (LCHAD), verylong-chain acyl-CoA dehydrogenase deficiency (VLCAD), infantile carnitine palmitoyl transferase deficiency type II (CPT II), carnitine-acylcarnitine translocase deficiency (CAT) and primary carnitine deficiency (PCD). Both CAT and (paradoxically) hepatic CPT I deficiency are particularly associated with sudden cardiac or respiratory arrest in the neonatal period<sup>11,12</sup>. Primary carnitine deficiency (PCD) is a systemic disorder resulting from a deficiency of the specific high-affinity carnitine transporter (OCTN2 transporter). The defect manifests acutely as hypoketotic hypoglycaemia with a Reye-like presentation but can also present as progressive cardiomyopathy or sudden death<sup>3,13,50</sup>. Biochemically, plasma and tissue carnitine levels are severely depleted. Pathologically, it may manifest as accumulation of lipid within skeletal muscle in type 1 myocytes, liver and frequently cardiac muscle. Electron microscopy (EM) may demonstrate abnormal mitochondria. Virtually all other carnitine deficiency states are secondary to associated conditions14.

#### **Diagnosis of Fatty Acid Oxidation Disorders**

Organic acid analysis will give clearly abnormal results in some but not all of these defects. Blood acylcarnitine profiles are reported to be more diagnostically specific and reliable than urine organic acid analysis but our experience is that acylcarnitine profiling also has its limitations. Ideally, both urine and blood should be analysed in order to obtain the maximum information. An acylcarnitine profile on post-mortem bile, ideally in conjunction with an acylcarnitine blood profile, can also provide useful complementary information<sup>3</sup>. Virtually all known primary longand medium-chain fatty acid oxidation disorders can be detected in cultured fibroblasts using the tritium release assays<sup>15,16</sup>. Acylcarnitine profiles generated by the incubation of labelled fatty acids with cultured fibroblasts are an alternative method for diagnosis<sup>17,18,52</sup>. Diagnosis can be confirmed by further specific enzyme assay,

complementation analysis in fibroblasts, or in the case of LCHAD by mutation analysis, the common E474Q LCHAD mutation accounting for about 87% of disease in Western Europe<sup>19</sup>.

Pathologically, there is fatty infiltration of the liver cells (macrovesicular and microvesicular steatosis), muscle cells and cardiac myocytes. Fat may also accumulate in the renal tubular cells and this may be more specific (but less sensitive) for defects of fatty acid metabolism than mild to moderate fat accumulation in other tissues. Some degree of fatty change, however, e.g., in the liver, may be a non-specific finding.

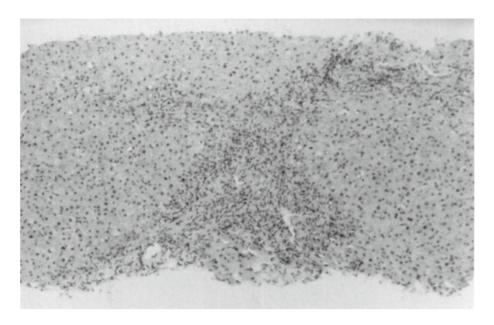
## **Urea Cycle Defects – Disorders of Ammonia Detoxification**

Defects of the Urea Cycle may present in the neonatal period, infancy or childhood or in adolescents and adults; cumulatively they have an incidence of 1:20,000. Hyperammonaemia is a potent cause of cerebral oedema. The presentation at different age groups is summarised below:

- Neonates show rapidly progressive symptoms often appearing on the second day
  of life with a short pre-symptomatic interval. Symptoms include poor feeding
  with lethargy, hyperventilation, seizures and progressive encephalopathy and
  coma. There is loss of reflexes and frequently intracranial haemorrhages resulting from coagulation defects. There is respiratory alkalosis with marked hyperammonaemia.
- 2. **Infants and children** present with failure to thrive, feeding problems, vomiting, chronic neurological symptoms, often as ataxia and seizures in association with metabolic decompensation. Patients can show rapid progression to encephalopathy and coma.
- Adolescents and adults show chronic neurological symptoms with psychiatric
  and behavioural problems. There may be lethargy, psychosis, disorientation and
  recurrent encephalopathy in association with high protein intake or catabolic
  stress.

Diagnosis, depending on the defect, is by amino acid analysis in plasma and urine, demonstration of elevated argininosuccinic acid in urine in the case of argininosuccinate lyase deficiency, orotic acid quantitation in urine followed by enzyme studies in leucocytes, fibroblasts or liver (where appropriate) and mutation studies.

The pathological changes in the neonate with ornithine transcobalamase (OTC) deficiency (x-linked disorder) may be non-specific, indeed the liver parenchyma and renal tubular epithelium may appear normal; however, in the hemizygous boy the liver may be enlarged with focal cellular necrosis and macro-/micro-steatosis (Fig 2.1). In older heterozygous girls, there may be piecemeal necrosis, non-specific inflammation, steatosis and fibrosis. At an ultrastructural level peroxisomal swelling and matrix rarefaction may be observed. The central nervous system (CNS) changes include Alzheimer type II astrocytes, particularly in the segmental areas of the pons and midbrain, with spongiosis, hypomyelination and cerebellar heterotopias. It is these latter changes which may be amenable to detection on magnetic resonance imagery (MRI). (P.D. Griffiths, personal communication).



**Fig. 2.1** Liver in OTC deficiency. Needle biopsy taken in life. There is hepatitis. The parenchyma shows finely vesicular pale staining with hyperpyknotic nuclei.

#### **Mitochondrial Respiratory Chain Disorders**

Mitochondriopathies are disorders of enzymes or enzyme complexes that are directly involved in the production of chemical energy by the process of oxidative phosphorylation. These include pyruvate dehydrogenase (PDH) complex and the respiratory chain complexes including ATP synthase. These disorders lead to a wide spectrum of clinical disease involving virtually all organ systems.

The ubiquitous nature of oxidative phosphorylation dictates that a defect of the mitochondrial respiratory chain should be considered in any patient presenting with an unexplained combination of neuromuscular and/or non-neuromuscular symptoms, with a progressive course, involving seemingly unrelated organs or tissues<sup>20</sup>.

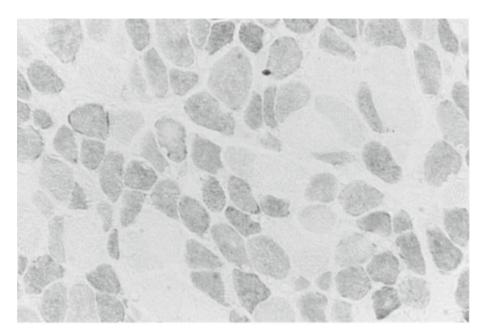
Almost any organ or system can be affected, but a combination of two or more of the following should alert suspicion. Neurological presentation with central and/or peripheral nervous system involvement. This may start in the neonatal period with drowsiness, poor sucking, severe hypotonia, respiratory distress, apnoeas and seizures often leading to fatal ketoacidotic coma with lactic acidosis. There may be a delayed onset with psychomotor regression, mental retardation with subacute brainstem necrosis and leucodystrophy. Muscular presentation ranges from fatal infantile myopathy to progressive muscle weakness in childhood. Cardiac disease in infants and older children is frequently a result of genetic defects of oxidative phosphorylation. This may present as an isolated cardiomyopathy or with other multi-organ involvement (see section on cardiomyopathy below).

Hepatic involvement either as a severe neonatal form or as delayed disease onset are also consequences of respiratory chain disorders. Histological examination is abnormal with steatosis, micro- and macronodular cirrhosis and elevated plasma and cerebral spinal fluid (CSF) lactate. Deficiency of complexes I and IV and multiple enzyme deficiency have been observed while mitochondrial DNA depletion has been implicated in a number of patients with severe hepatic failure. Renal disease presenting as proximal tubulopathy with De Toni-Debre-Fanconi syndrome or glomerular disease with nephrotic syndrome and chronic tubulointerstitial nephropathy are again features of respiratory chain disease. Renal biopsy shows non-specific abnormalities of the tubular epithelium with dilations or obliteration by casts, atrophy or dedifferentiation, occasionally with giant mitochondria. Intrauterine growth retardation, spontaneous abortion and intrauterine death are also associated with respiratory chain disease. Alternatively, there may be postnatal growth failure at any age, even after several months of apparently normal development. Recurrent vomiting, severe anorexia, chronic diarrhoea with villous atrophy, and/or exocrine pancreatic dysfunction may occasionally occur. Disturbance of endocrine function may also occur presenting as dwarfism, diabetes mellitus, hypoparathyroidism and, rarely, hypothyroidism and ACTH deficiency. Other affected tissues include haemopoietic tissue with refractory anaemia, neutropenia and thrombopenia, e.g., Pearson syndrome, various dermatological problems affecting skin and hair and dysmorphology with facial anomalies, microcephaly, and involvement of the limbs and trunk with short hands, brachydactyly and hypoplasia of the distal and middle phalanges.

#### **Diagnosis of Mitochondrial Respiratory Chain Defects**

Diagnosis of respiratory chain disorders is particularly problematic not least because the disease may have variable tissue expression, may be partial and involve several respiratory chain components. Measurement of plasma and/or CSF lactate and lactate/pyruvate ratios are useful investigations only in living patients. The respiratory chain complexes are particularly labile, exhibiting rapid deterioration after death and in biopsied material unless snap frozen and stored at -80 °C, ubiquinone being particularly labile giving rise to low complex II/III activity (I.P. Hargreaves, personal communication). The complexes are also prone to undergo secondary loss of function in vivo, in particular as a result of reperfusion following tissue anoxia<sup>21</sup>. Most investigations are therefore only informative in living patients, the exception being analysis of mitochondrial DNA (provided the correct tissue is biopsied) and nuclear encoded mutations, a number of which have now been described. The measurement of the respiratory chain complexes in postmortem tissue may be informative in samples taken up to two hours after death, but wherever possible, a biopsy taken prior to death will give more reliable information. Most respiratory chain disorders presenting in neonates or children below five years of age are nuclear encoded defects, although they may show variable tissue expression. Conversely, mitochondrial DNA gene defects that are seen in older children and adults with respiratory chain disease, e.g., myoclonic epilepsy with ragged red fibres (MERRF), encephalomyopathy, lactic acidosis, stroke-like episodes (MELAS), are rare in young children.

Organic acid analysis of urine may give abnormal findings with increases in 3-methylglutaconic acid, dicarboxylic and 3-hydroxydicarboxylic acids, Kreb's cycle intermediates, e.g., fumarate, malate, succinate and increased excretion of lactate and ketones, however, all these metabolites are non-specific and can only serve as pointers to possible diagnosis.



**Fig. 2.2** Muscle in mitochondrial respiratory chain disease. Complex IV (COX) staining shows some COX-negative fibres.

Measurement of respiratory chain complexes I, II, III, IV and ubiquinone in freshly frozen muscle by spectrophotometric assay and/or measurement of mitochondrial function by polarography/high-resolution respirometry in fresh muscle or isolated muscle mitochondria are considered to be the gold standard for the detection/diagnosis of respiratory chain disease<sup>28–32</sup>. However, these highly specialised assays are available in only a few centres and are dependent on fresh muscle being analysed without delay or on fresh, snap-frozen muscle stored and transported at very low temperature.

A significant proportion of neonates and young children presenting with respiratory chain disease express the defect in cultured fibroblasts<sup>22–35</sup>. In our experience, cultured fibroblasts from many of these patients show reduced fatty acid oxidation flux as a secondary consequence of a block in the respiratory chain. Alternatively, provided that the defect is expressed in fibroblasts, digitised cultured fibroblasts can be used to demonstrate decreased ATP production with a variety of substrates<sup>26,27</sup>. Spectrophotometric assays for complexes II, III, IV and ubiquinone in fibroblasts can, as in muscle, be used to confirm deficiency.

Electron microscopy reveals a number of mitochondrial alterations in mitochondriopathies. The number and size of the mitochondria are often increased. Giant mitochondria with concentric lamellar tubular reticular or dissociated cristae are characteristic. The mitochondrial matrix may be swollen and display large spherical dense bodies, vacuoles or crystals. Rectangular crystals may be arranged in blocks of parallel crystals with a "parking lot" configuration. Light microscopy and EM investigation show changes which are not necessarily characteristic. Ragged red fibres (RRF) may be seen in a small proportion of muscle fibres in neuromuscular disorders other than in mitochondriopathies but RRF are rarely seen

in children with respiratory chain disease. Zidovudine may also cause RRF. Histochemical staining may show absent or weak cytochrome oxidase activity (COX) in some mitochondrial complex IV (cytochrome oxidase) defects (Fig 2.2). However, we have experienced false-positive staining for COX in patients who show very low complex IV activity when measured spectrophotometrically in fresh muscle. Moderately increased fat droplets and glycogen pooling may also be seen, occasionally in association with RRF, but extensive infiltration of fat is more characteristic of disorders of fatty acid oxidation.

In our experience, in many cases of suspected respiratory chain disease the cumulative evidence from the family history together with the available clinical, histological and biochemical/molecular information needs to be carefully considered before attributing a degree of probability to the diagnosis.

#### **Organic Acidurias**

This large group of disorders results from defects of intermediary metabolism with the characteristic accumulation of carboxylic acids in urine. The majority of the important organic acidurias are caused by disorders involving the metabolism of branch chain amino acids. Patients classically present in the neonatal period with metabolic encephalopathy resulting from "metabolic intoxication". There is lethargy, feeding problems, dehydration, limb hypertonia with trunkal hypotonia. Patients show multi-system failure, neurovegetative dysregulation and coma. These disorders include isovaleric aciduria (IVA), propionic aciduria (PA), methylmalonic aciduria (MMA) and multiple carboxylase deficiency. The multiple carboxylases are biotin-dependent enzymes important in the metabolism of branched chain amino acids, and disorders of these enzymes encompass biotinidase deficiency, holocarboxylase synthetase deficiency and 3-methylcrotonylglycinuria. Chronic intermittent and chronic progressive forms of these disorders occur. These tend to present later, either in infancy but also manifesting up to adulthood with recurrent episodes of ketoacidotic coma, lethargy and ataxia sometimes resulting in coma with Reye-like presentation or with chronic failure to thrive, episodes of vomiting, anorexia, osteoporosis, hypotonia and developmental retardation. Glutaric aciduria type I (GA I) is a disorder of lysine and tryptophan metabolism. Patients are normal at birth but usually present during infancy or early childhood with acute encephalopathic crisis and metabolic decompensation during intercurrent infection or other metabolic stress. There is often macrocephaly with frontotemporal atrophy. The first episode may be fatal but those patients that survive are left with severe dystonic-dyskinetic movement disorder; intellect, however, is frequently intact. Most patients excrete significant quantities of glutaric acid and 3-hydoxyglutarate in the urine and have low plasma carnitine levels. A small but significant percentage of patients may give false-negative results on organic acid screening and definitive diagnosis is by enzyme assay in fibroblasts or fresh lymphocytes. Patients presenting with this condition may be mistaken as cases of possible non-accidental injury (refer to later section on NAI).

#### 3-Hydroxy-3-methylglutaryl (HMG) CoA Lyase

This is an enzyme required for ketogenesis as well as for the last step in leucine oxidation. Patients with deficiency of this enzyme present in infancy or early child-

hood with life-threatening, Reye-like crisis; the picture is one of hypoketotic hypoglycaemia, metabolic acidosis and liver disease. Analysis of urine organic acids gives a specific pattern of abnormal metabolites.

## 3-Ketothiolase (Mitochondrial 2-methylacetoacetyl-CoA Thiolase)

This is a ketolytic enzyme but is also an enzyme of isoleucine metabolism. Patients usually present in infancy, usually between 6 and 24 months of age, but occasionally as older children, with episodes of vomiting often precipitated by intercurrent infection or occasionally by high protein intake. There is acute life-threatening ketoacidotic crisis with severe metabolic acidosis, occasionally low, but usually normal or high blood glucose and normal or moderately raised ammonia. Patients may rapidly succumb to overwhelming metabolic acidosis. Urine organic acid and blood acylcarnitine analysis will identify this disorder.

## Multiple Acyl-CoA Dehydrogenase Deficiency (MADD or Glutaric Aciduria Type II – GA II)

This is a not only a defect of fatty acid oxidation but of several other dehydrogenases involved in the metabolism of branched-chain amino acids, lysine and sarcosine. There is a spectrum of clinical phenotypes depending on the level of residual activity. The most severe form shows congenital abnormalities, the most notable being renal dysplasia and multiple cysts, but there may also be facial dysmorphism, rocker-bottom feet, hypospadias and chordee. Severe forms usually succumb during the first week of life. The less-severe neonatal/infantile form tends to manifest somewhat later with hypoketotic hypoglycaemia, metabolic acidosis, progressive encephalopathy, epilepsy and cardiomyopathy. Milder forms may present at any time from infancy to adulthood with hypoketotic hypoglycaemia and acidosis and may progress to encephalopathy and coma. All forms show characteristic organic acid abnormalities in crisis, blood acylcarnitines also giving clearly abnormal results.

In the broader context organic acidaemias also include many of the fatty acid oxidation disorders.

#### **Disorders of Amino Acid Metabolism**

This varied group of disorders results from deficiencies in the enzymes involved in amino acid metabolism, frequently resulting in accumulation of toxic substances leading to organ damage. Classically, they are recognised by amino acid analysis in plasma or urine and do not fundamentally differ from the organic acidurias that are diagnosed by organic acid analysis. They may present in a similar manner to a number of organic acidaemias with acute decompensation involving the brain, liver and kidney, tyrosinaemia type I (Fig 2.3), sulphite oxidase deficiency and molybdenum cofactor deficiency are examples. Diagnosis is by analysis of plasma and urine amino acids, urinary succinylacetone in the case of tyrosinaemia type I, demonstration of sulphite in fresh urine in sulphite oxidase deficiency or increased urinary xanthine and hypoxanthine in molybdenum cofactor deficiency.

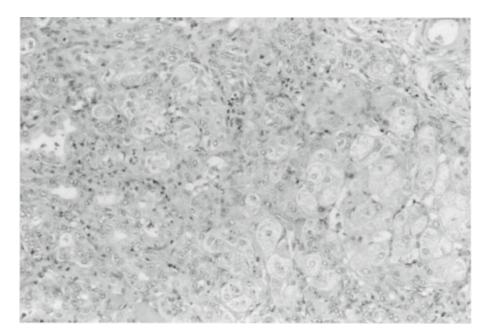


Fig. 2.3 Liver in tyrosinaemia type I. Note the intense fibrosis "nesting" around groups of liver cells.

#### Maple Syrup Urine Disease (MSUD)

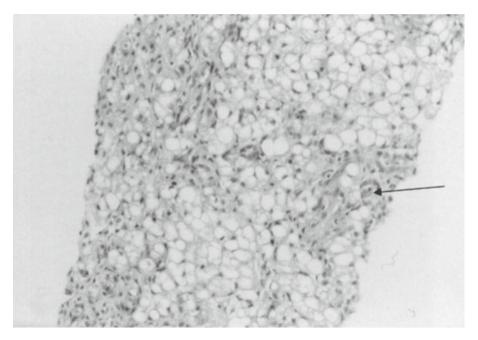
This is a disorder of branched-chain amino acid metabolism. It classically presents as progressive encephalopathy after 3 to 5 days of life, but a milder intermittent form presenting later in infancy or childhood is also well described. There is initial lethargy with poor feeding which progresses to encephalopathic coma. In contrast to classical organic acidurias, acidosis and hyperammonaemia are not major features of the disease. Plasma levels of branched chain amino acids are grossly elevated, particularly leucine and the urine contains branch-chain keto- and hydroxy-acids.

#### **Disorders of Galactose and Fructose Metabolism**

Patients with disorders of galactose or fructose metabolism only develop clinical symptoms of disease after ingestion of lactose (milk and dairy products) or fructose/sucrose respectively. Galactose-1-phosphate and fructose-1-phosphate, which accumulate in classical galactosaemia and hereditary fructose intolerance, are toxic metabolites that lead to organ damage, particularly in liver, kidneys and brain. Galactosaemia usually presents in the neonatal period on commencement of milk feeds and is frequently fatal unless detected at an early stage. Hypoglycaemia with jaundice, deranged liver function and renal failure are the common features often with accompanying sepsis. Patients may succumb with the incorrect primary diagnosis of sepsis. The pathologic changes include a marked steatosis of the hepatocytes with progressive pseudoacinar change and ductular proliferation, cholestasis,

focal necrosis and eventual cirrhosis (Fig 2.4). Pancreatic islet cell hyperplasia and vacuolation of the renal tubular epithelium also occurs. The changes seen are similar to those observed in hereditary fructose intolerance (see below).

Within the CNS at autopsy, there may be oedema, gliosis and neuronal necrosis, all attributable to hypoxic-ischaemic damage. The foetus may show evidence of incipient cataracts. Diagnosis for galactosaemia is by enzyme assay in whole blood or quantitation of galactose-1-phosphate in erythrocytes, or by mutation detection. Blood transfusion will, of course, invalidate the enzyme assay in blood.



**Fig. 2.4** Liver in galactosaemia showing macrovesicular steatosis and fibrosis. Note the bile plug in the bile duct (arrow).

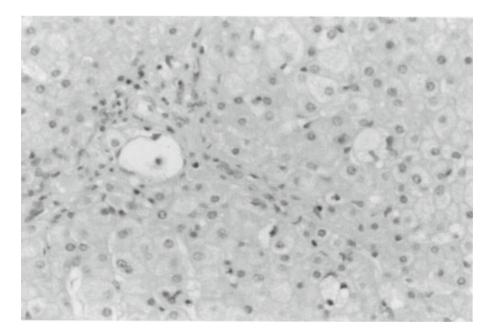
#### **Hereditary Fructose Intolerance**

This may present with similar features to galactosaemia at the time of weaning with the first introduction of fructose-containing feeds. Patients present with hypogly-caemia, vomiting, progressive liver dysfunction with hepatomegaly, renal tubular damage and coma. Diagnosis is by demonstration of reducing substance in the urine (fructose) and often by demonstrating the presence of the common A149P mutation in the aldolase B gene. Galactosaemia and hereditary fructose intolerance show similar pathologic changes, e.g., fatty change in the liver, giant cell transformation, pseudoacinar arrangement of hepatocytes and cirrhosis.

#### **Lysosomal Storage Disorders**

Some 40 different lysosomal disorders are described in humans. These disorders result from a defective function of a specific protein which ultimately leads to the

progressive accumulation within the lysosome of either undegraded substrate or catabolic products that are unable to escape from the lysosome<sup>33</sup>. Most of these disorders are inherited in an autosomal recessive manner. The accumulation of storage material within the lysosome is generally progressive and unremitting and as a consequence the clinical phenotype, although remarkably varied, tends to follow a chronic degenerative course. However, some disorders may present in utero or in the neonatal period as hydrops fetalis or as neonatal death, e.g., mucopolysaccharidosis type IVb, mucopolysaccharidosis type VII, Niemann-Pick type C, GM<sub>1</sub>-gangliosidosis mucolipidosis type I, II and galactosialidosis<sup>34</sup>, whereas in others, even with the same enzyme deficiency, onset of symptoms may be in late adulthood. For most patients, however, the onset of symptoms will be in the first months or years of life often following an apparently normal early development. The first signs may be slowed development with other neurological signs; in other patients some visceromegaly or coarse features may be noted. The clinical phenotype is often a guide in the selection of appropriate diagnostic tests. Testing for many of these disorders in life is by enzyme assay in fresh white blood cells and/or in the case of mucopolysaccharidoses (MPS) and oligosaccharidoses by detecting the excessive excretion of glycosaminoglycans and oligosaccharides in urine, respectively. The pathological changes of GM<sub>I</sub>-gangliosidosis vary with type and age of onset but in all cases there is visceromegaly with laminated inclusions (ganglioside and/mucopolysaccharide) within reticuloendothelial cells, liver cells, renal glomerular and tubular epithelium, pancreatic and mucoserous gland epithelium, central and peripheral neurones. Similarly, there is sphingomyelin and cholesterol accumulation in foamy cells of the reticuloendothelial system in Niemann-Pick type C (Fig 2.5).



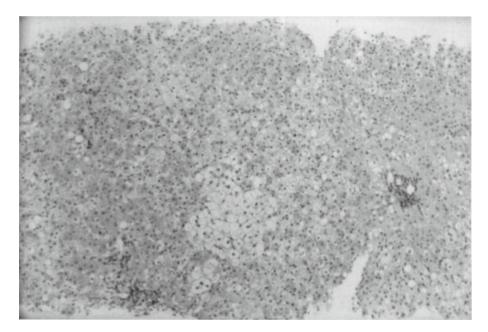
**Fig. 2.5** Liver in Niemann-Pick type C – scattered cells with vesicular cytoplasm due to cholesterol accumulation. There is inflammatory infiltration around the hepatic vessel.

More generally, material stored in the various types of affected cells includes lipid, sphingolipids, sulphatides, phosphosphingolipids, lipofuscins, polysaccharides and mucopolysaccharides and lipoproteins, and many of these can be demonstrated by techniques of histochemical staining and electron microscopy. Detection of characteristic cells in bone marrow or vacuolated lymphocytes may also confirm or suggest the diagnosis. Enzyme studies can also be performed in cultured fibroblasts.

## **Glycogen Storage Disorders**

Glycogen storage disease (GSD) presents invariably either as pathological accumulation of glycogen (e.g., isolated hepatomegaly) with corresponding organ dysfunction (e.g., liver disease and/or myopathy or with hypoglycaemia).

The enzyme defect is frequently organ specific and therefore there may be primary hepatopathic (types I, IIIb, IV, VI, IX) (Fig 2.6), myopathic (types V, VIII) or mixed (types II, IIIa) symptoms. The cumulative incidence of GSD is 1:20,000, all except type VI (x-linked) is autosomal recessive. Patients may present in early infancy with hypotonia and severe cardiomyopathy (type II – Pompe) or with hypoglycaemic seizures, recurrent hypoglycaemia with acidosis, trunkal obesity, hepatomegaly, nephromegaly, muscle atrophy and bleeding tendency (type I) or progressive liver disease with cirrhosis and splenomegaly (type IV). There may be a more progressive myopathic presentation in children or young adults with primarily muscle weakness (mild type II or type V and VII). Diagnosis is confirmed by biopsy/enzyme studies or mutation analysis. At a microscopic level most show



**Fig. 2.6** Liver in glycogen storage type la. There is steatosis with a few inflammatory cells. There is marked hydropic change in hepatocytes reflecting glycogen accumulation. There is some inflammation around the portal tract.

massive accumulation of glycogen in the affected tissue(s), a notable exception being glycogen synthase deficiency where the finding is a marked reduction in liver glycogen.

### **Peroxisomal Disorders**

These is a group of genetically determined disorders of either peroxisomal biogenesis or of a single peroxisomal protein<sup>35</sup>. This group may broadly be divided in two:

- 1. Failure to form or maintain the organelle resulting in defective function of multiple enzymes.
- 2. Genetically determined defect of a single peroxisomal enzyme or protein with retained integrity of the peroxisome.

The peroxisome has a wide range of important functions including the  $\beta$ -oxidation of very-long-chain fatty acids and related compounds as well as synthesis of plasmalogens, cholesterol and bile acids. It also has a key role in protecting the cell against free radical damage. As a consequence of the many key functions, many peroxisomal disorders present in the neonatal period and may be rapidly fatal. Features of peroxisomal disease include:

- 1. Dysmorphic features particularly craniofacial abnormalities.
- 2. Skeletal abnormalities in particular short proximal limbs and calcific stippling.
- 3. **Neurological abnormalities** with encephalopathy, hypotonia, deafness and seizures.
- 4. Eye abnormalities retinopathy, cataracts and blindness.
- 5. Hepatointestinal dysfunction cirrhosis, cholestasis and hepatomegaly.

The measurement of very-long-chain fatty acids, phytanic acid, pristanic acid and bile acids in plasma and plasmalogens in erythrocytes will detect all peroxisomal defects but further studies usually in fibroblasts may be necessary for accurate characterisation of the defect. Immunocytochemical methods in various tissues, particularly liver and kidney<sup>36</sup>, or peroxisomal morphometry by electron microscopy are alternative methods for diagnosis at autopsy<sup>37</sup>. An example of this is in Zellweger syndrome where there are striking abnormalities in the brain characterised by disorder of neuronal migration and involving the cerebral hemispheres, cerebellum and inferior olivary nucleus. Brain abnormalities include lissencephaly, and other cerebral gyral disorders, heterotopic cerebral cortex, olivary nucleus dysplasia, defects of the corpus callosum, numerous lipid-laden macrophages, histiocytes in cortical and periventricular areas and dysmyelination. Electron microscopy reveals an absence of peroxisomes in the liver and kidney and this is also demonstrable in cultured fibroblasts and amniocytes.

## **Post-mortem Findings**

The post-mortem investigation should begin with evaluation of clinical notes, x-rays and assessment of any pre-mortem biochemical investigations. Features that may suggest a metabolic cause of death are outlined in Table 2.1.

The external examination must include careful evaluation of any dysmorphic features. Table 2.2 shows those features associated with specific disorders. In the future some of the subtle internal changes, e.g., cerebral dysgenesis, agenesis of the corpus callosum and congenital heart anomalies, may be amenable to detection by post-mortem MRI.

In cases of early neonatal death, assessment of the placenta may also yield vital information, e.g., vacuolation of the syncytiotrophoblast may arise in a number of storage disorders. Newer techniques such as MRI may allow targeted biopsies in cases with limited consent. In many of the disorders suspicion may be aroused by microvesicular steatosis of the liver and/or renal tubule and coarse vacuolation of renal tubular epithelium. Storage material in various organs and/or tissues may offer vital clues to a diagnosis.

## **Specimens at Post-mortem**

The collection of appropriate samples at post-mortem is of paramount importance in the investigation of metabolic disease, for in the absence of appropriate samples accurate diagnosis is impossible, and informed genetic counselling for the family, and reliable risk assessment for future children cannot be offered. It is essential to collect representative post-mortem samples and to discuss their analysis with a metabolic specialist. Appropriate investigations on often very small samples of body fluids and tissues must be well coordinated if precious material is not to be used up unnecessarily. In cases of expected death, whenever possible, collect samples of blood, urine and tissues prior to death. This applies especially to skeletal muscle biopsies and CSF for the investigation of suspected mitochondrial respiratory chain disease, as samples collected after death are highly susceptible to post-mortem deterioration. It is also of paramount importance to obtain a skin biopsy for fibroblast

**Table 2.1** Abnormalities suggesting underlying metabolic disorder.

Neurologic	Eyes	Skin	Gastro- intestinal	Musculo- skeletal	Other
Coma	Cataract, Corneal clouding	Eczema	Poor feeding	Myopathy	Dysmorphic features
Hypo-/ hypertonia	Cherry-red macula	Angiokeratoma	Recurrent vomiting	Arthritis	Neonatal death
Lethargy	Dislocated lens	Photosensitivity	Jaundice	Abnormal movements	Consanguinity
"Fits"	Glaucoma	Xanthoma	Failure to thrive	Spasms	Abnormal body odour
Movement disorders	Retinitis	Oedema	Hepato-megaly		Abnormal hair texture
Developmental delay			Hydrops ascites		Splenomegaly, cardiomegaly
Progressive psychomotor deterioration					Recurrent acidosis +/- ketosis

Modified from Barness L., Gilbert-Barness E. Metabolic Diseases In: Gilbert-Barness E, editor. Potter's pathology of the fetus and infant, St Loius: Mosby Year Book, 1997, Chapter 17 (Vol. 1): 572.

**Table 2.2** Dysmorphic features associated with metabolic disorders. Modified from Dimmick JE, Kalousek DK. Developmental pathology of the embryo and fetus. Philadelphia: JB Lippincott, 1992.

Phenotype	Metabolic Disease		
Hydrops fetalis	Wide variety of metabolic disorders including lysosomal storage		
disorders			
Facial dysmorphism			
Genital anomalies			
Congenital heart disease*			
Renal cystic dysplasia			
Glomerulopathy			
Cerebral dysgenesis*	Glutaric aciduria type II		
Microcephaly	··		
"Foetal alcohol" facies			
Agenesis of the corpus callosum*	Pyruvate dehydrogenase		
Facial dysmorphism	, , ,		
Epiphyseal calcification			
Congenital heart disease			
Renal microcysts			
Cerebral dysgenesis	Zellweger syndrome		
Facial dysmorphism	,		
Hepatopathy	Infantile Refsum disease		
Facial dysmorphism			
Rhizomelic limb shortening	Rhizomelic chondrodysplasia punctata		
Frontal bossing			
Low-set ears	GM <sub>i</sub> -gangliosidosis		
Ambiguous genitalia	Congenital adrenal hyperplasia		
Coarse facial features	3		
Stippled epiphysis	Sialidosis		
Coarse facial features	Mucolipidosis type II		
Coarse facial features	Mucopolysaccharidoses		
Coarse facial features	Infantile sialic acid storage disease		
Congenital heart defects*			
Agenesis of the corpus callosum*	β-Hydroxyisobutyryl-CoA deacylase deficiency		
Arthrogryposis	Gaucher-like storage disease		

<sup>\*</sup>These changes may be amenable to detection by post-mortem MRI.

culture, as many of the enzymes known to be deficient in inborn errors of metabolism are expressed in skin fibroblasts, and this will frequently be the only tissue where an enzyme confirmation of a suspected diagnosis can be made. Cultured fibroblasts also provide important reference material for future prenatal diagnosis for the family concerned as these can be cryopreserved for an indefinite period in liquid nitrogen.

## Samples

#### Urine

This should be collected by catheterisation or suprapubic puncture into a container with no preservative; as little as  $100~\mu l$  can be sufficient for organic acid analysis by gas chromatography mass spectrometry (GC-MS). If the sample is contaminated with blood, centrifuge the sample to remove the cells prior to freezing the urine at

-20 °C. Washing out the bladder with a small volume of sterile saline may also yield enough sample for organic acid analysis.

#### Blood

If only small quantities of blood are available, whole blood acylcarnitine analysis is likely to be the single most informative test that can be performed on blood collected at post-mortem in spite of the potential problems of interpretation<sup>3</sup> (see Basic Investigations). Spot a few drops of whole blood onto a Guthrie card as this is the most convenient method of collection. Whole blood is in our experience preferable to plasma as the acylcarnitine profile tends to have lower background interference. Long-chain acylcarnitines also tend to stick to red blood cells so can be underrepresented in the plasma profile. If more blood is available (in addition to the Guthrie card) collect up to 10 ml of heparinised blood as this can be separated and the plasma stored at –20 °C, while the cells should be stored at +4 °C (do not freeze). If DNA analysis is likely to be required collect a further 5 ml of whole blood (EDTA) and freeze immediately (at least –20 °C) until DNA is required. Where blood can be obtained before or very soon after death also collect 5 ml of blood into a fluoride oxalate tube.

## **Cerebral Spinal Fluid**

Cerebral spinal fluid may be useful in certain circumstances. Collect two 1 ml samples, one into a plain tube and one with fluoride oxalate and store these at -80 °C.

### **Vitreous Humour**

This can be collected into a fluoride bottle by needle aspiration and stored at -20 °C.

#### Bile

Bile can be collected at post-mortem into a plain tube for storage at -20 °C or alternatively spotted onto a Guthrie card for acylcarnitine profiling.

### Skin

Sterility is of paramount importance when taking a skin biopsy. Small biopsies carry a lower risk of infection. Two 3 mm  $\times$  3 mm full-thickness biopsies are all that is required; alternatively, two punch biopsies taken from different sites will suffice. Large pieces of skin and tissue frequently fail because of infection. It is recommended that the skin biopsy is taken at the beginning of the post-mortem to reduce the risk of contamination. A skin biopsy taken up to 48 hours post-mortem is likely to be viable provided it is not infected. However, the biopsy should be obtained as soon as possible after death as this increases the likelihood of a successful culture. Because of the high risk of infection for post-mortem skin biopsies it is advisable to take two separate biopsies from different sites and to put the biopsies in two separate containers of special sterile culture medium. The addition of Fungizone to the culture medium can help to reduce the risk of fungal infections, but it is no

substitute for good aseptic technique. Once the biopsies have been taken they should be sent immediately to the cell culture laboratory, but can be stored overnight, or exceptionally for up to 36 hours at +4 °C (do not freeze) prior to dispatch. In an emergency, sterile normal saline can be used instead of culture medium, but do not use agar.

## **Tissue Samples**

The acquisition of organ biopsies depends on the clinical picture and is best discussed with a metabolic specialist. Liver, heart muscle, skeletal muscle and kidney are only suitable for biochemical analysis if taken within 2 hours of death. Open biopsies are preferable, but if this is not possible, two or three needle biopsies should be taken. All such biopsies for biochemical analysis should be wrapped in aluminium foil and snap frozen in liquid nitrogen or solid carbon dioxide. The samples should then be stored deep-frozen in a -80 °C freezer.

Tissue samples for histological examination:

#### Liver biopsy

- Piece in formalin for histology.
- Small piece into glutaraldehyde for electron microscopy.

#### Muscle biopsy

- Small piece for formalin fixation and paraffin preparation (do not freeze) for light microscopy.
- Thin muscle fibre, tied to a stick (to avoid contraction and preserve orientation) in 2% glutaraldehyde on ice, for electron microscopy.

## **Investigations and Analysis**

The investigation of suspected metabolic disease when there is sudden death of unknown cause, or where investigations in life have not resulted in a diagnosis, requires a coordinated plan of action. It is important to obtain accurate clinical and family histories, e.g., ethnicity, consanguinity, together with any pre-mortem biochemical information and preliminary post-mortem findings and use this information to dictate the direction of further investigations.

## **Basic Investigations**

## Amino Acids in Plasma, CSF and Urine

Aminoacidopathies and urea cycle defects will give abnormal profiles in samples taken prior to death but interpretation of post-mortem material is often problematic. Most suspected defects of amino acid metabolism will require enzyme/biochemical confirmation in the appropriate tissue in order to confirm a diagnosis. It is worth noting that some of the urea cycle defects can only be enzymatically confirmed on fresh liver biopsy, i.e., ornithine transcarbamylase (OTC) deficiency or in the case of carbamylphosphate synthase 1 (CPS 1) deficiency in fresh liver or colon. Mutation analysis circumvents the need for liver biopsy in these disorders where a specific diagnosis is strongly suspected.

## Organic Acids in Urine

Organic acidurias, including many of the fatty acid oxidation defects and a significant number of other defects of intermediary metabolism, will give abnormal organic acid results; some profiles may in themselves be pathognomonic, as in the case of MMA, classical IVA, GA I or MCAD, but other findings may be less specific and highlight the need for further investigations.

## Acylcarnitine Profile on Guthrie Card Blood Spot

Acylcarnitine profile will detect many inherited metabolic defects including most fatty acid oxidation disorders and some organic acidaemias<sup>3</sup>. However, few of the profiles from post-mortem blood are in themselves reliably diagnostic and will need further tests, often on fibroblasts, to confirm a diagnosis. Post-mortem changes can make interpretation of acylcarnitines especially problematic, and it is often worth seeking out the stored six-day neonatal screening card (if this is available) for acylcarnitine analysis, as this is potentially more reliably informative.

### Cis-4-decenoic Acid in Plasma

This compound is significantly raised in cases of MCAD and although not entirely specific for MCAD, it serves as a very useful indicator of this disorder.

#### **Further Tests**

## **Total and Free Carnitine Analysis**

Analysis by tandem-MS can be performed on as little as 70  $\mu$ l of plasma and can be particularly informative in the differential diagnosis of fatty-acid oxidation defects. Both total and free carnitine are low in many organic acidaemias, e.g., GA I, and may serve as a useful indicator of an underlying metabolic defect. A high acylcarnitine fraction with very low free carnitine is also indicative of some metabolic disorders, e.g., CPT II, CAT. However, blood carnitine levels rise fairly rapidly after death and therefore blood samples need to be taken soon after death and even then results need to be interpreted with caution.

## Intermediary Metabolite Profile

This profile, consisting of glucose, lactate, free fatty acids and 3-hydoxybutyrate, can be performed on "fluoride" plasma. Results can be very informative provided the sample was collected prior to death.

# Very-long-chain Fatty Acids (VLCFA's), Phytanic and Pristanic Acid and Bile Acids

These compounds can be measured in plasma or serum by GC-MS in cases of suspected peroxisomal disorders. Almost all known peroxisomal defects can be detected if all four parameters are measured.

#### **Vitreous Humour**

Organic acid analysis of vitreous humour may be useful in the absence of urine, e.g., 7-hydroxyoctanoate is elevated in cases of MCAD. Vitreous humour glucose, provided the sample was taken peri-mortem into a fluoride tube, will reflect plasma glucose levels at that time, e.g., a normal glucose result in an acute admission (provided the patient was not administered glucose prior to death) excludes hypoglycaemia as a cause of collapse.

#### **Bile**

Acylcarnitine and bile acid profiles can be measured in small quantities of postmortem bile but need careful interpretation and require supporting biochemical evidence for accurate diagnosis. Bile may be especially useful if there is no blood available for acylcarnitine analysis but it is likely to prove more informative when interpreted in conjunction with a blood spot acylcarnitine profile<sup>3</sup>.

## **Molecular Genetic Investigations**

Mutation analysis may be used to confirm a diagnosis where enzyme confirmation is not possible, e.g., organ-specific disease expression (ornithine transcarbamylase (OTC), mtDNA from affected tissue in respiratory chain disorders), disorders of structural, receptor or membrane proteins (sarcomeric proteins in isolated cardiomyopathy, osteogenesis imperfecta in severe neonatal bone disease) or in diseases with single common mutations (MCAD, long-chain 3-hydroxyacyl-CoA dehydrogenase deficiency (LCHAD)).

The preferred option for antenatal diagnosis for future pregnancies is frequently by mutation analysis on direct chorionic villus biopsy where this is possible. DNA for molecular analysis can be most easily obtained from EDTA whole blood. Guthrie card blood spots, frozen tissue biopsies and fibroblasts will also yield DNA. If RNA is required this can be obtained from cultured fibroblasts.

## **Tissue Assays**

## **Cultured Fibroblasts**

Many of the enzymes known to be deficient in inborn errors of metabolism are expressed in skin fibroblasts with the notable exception of some urea-cycle and glycogen-storage enzymes.

Homogenates or sonicates of cultured fibroblasts are suitable for many specific enzyme assays, or alternatively in some instances, assays using crude mitochondrial preparations may be more appropriate, e.g., the investigation of respiratory chain disorders<sup>20</sup>. One particular advantage of using intact living cells is that the uptake or incorporation of various substrates into the cell or the flux of metabolites through an entire pathway can be measured<sup>18</sup>. Additionally, cultured cells have the advantage that they are not subject to deterioration of enzyme activity or secondary loss of function that is so frequently a problem in biopsies and particularly other post-mortem samples. An example of the use of intact cells is in the diagnosis of fatty-acid oxidation by tritiated release assay in cultured fibroblasts<sup>38</sup>. More

recently, cultured fibroblasts have been incubated with deuterium-labelled fatty acids or other suitable substrates and the resultant acylcarnitine profiles analysed by tandem MS<sup>17</sup>. This technique has proved to be particularly informative in fatty acid oxidation disorders, but also has the potential for use in a wide range of other inherited metabolic diseases.

### **Other Tissues**

Fresh frozen muscle is often the tissue of choice for the diagnosis of mitochondrial respiratory chain disorders. Provided the muscle has been collected prior to death (or within two hours of death) complexes I, II, III and IV of the respiratory chain can be measured. Other fresh tissue such as heart and liver can also be used where specific involvement of these tissues is indicated, e.g., mutation analysis of cardiac mitochondrial DNA in the case of isolated cardiomyopathy or specific enzyme assay in liver for some urea cycle disorders.

### **Acute Metabolic Disease in the Neonatal Period**

Many inborn errors of metabolism can present in the newborn period. Most babies are born apparently healthy and may show a typical asymptomatic period with clinical manifestation from the second day of life onwards. However, some disorders may present at birth or may be detected by antenatal ultrasonography. Sadly, many babies with inherited metabolic disease follow a rapid course of deterioration and may succumb before appropriate investigations can be undertaken. The pattern of deterioration may be a useful indicator of the underlying disorder; see Table 2.3<sup>39</sup>.

## **Sudden Infant Death Syndrome**

Sudden Infant Death Syndrome (SIDS) is the unexpected death of an apparently well infant over one month of age, for which no cause can be found, in spite of a post-mortem examination<sup>40</sup>. The diagnosis of SIDS or sudden unexplained death in infancy (SUDI) still remains the largest single cause of death in children in the industrialised world. The frequency is reported at 1:1,000 live births and represents 25% of all deaths in the first year of life. It may result from dramatic cardiac failure, shock or cardiac arrest in many metabolic circumstances, particularly in defects of mitochondrial fatty acid oxidation and respiratory chain disorders. Professor John Emery was the first to observe that a wide range of metabolic disorders may present as sudden infant death syndrome<sup>41</sup>.

Though critical review may suggest that not all these deaths were entirely unexpected, they were initially unexplained. Identification of the cause of death in such cases is some consolation to parents and offers the opportunity for future prenatal diagnosis. Although at least 31 metabolic disorders are listed as causes of SIDS, there is some doubt as to the validity of some reports<sup>42</sup>. The most likely causes of sudden unexplained death are listed below:

- 1. Inherited defects of fatty acid oxidation and ketogenesis.
- 2. Urea cycle disorders most commonly OTC.
- 3. Organic acidurias, e.g., methylmalonic, propionic and isovaleric aciduria.

Liver disease	Acid-base disorders	Cardiac disorders	Neurological deterioration	Hypoglycaemia
Galactosaemia $\alpha$ -1-antitrypsin deficiency	Metabolic acidosis Organic acidaemias	Disorders of fatty acid oxidation Respiratory chain disorders	Hyperammonaemia Organic acidaemias	Disorders of fatty acid oxidation
Respiratory chain disorders	Congenital lacticacidaemias	Congenital disorders of glycosylation	Maple syrup urine disease	Fructose 1,6- bisphosphatase deficiency
Neonatal haemochromatosis	Fructose 1,6- bisphosphatase deficiency		Disorders of fatty acid oxidation	Glycogen storage disease type 1
Disorders of fatty acid oxidation Tyrosinaemia type 1	Ketolysis defects	Pompe disease	Congenital lacticacidaemias	Respiratory chain disorders
Niemann-Pick type C	Respiratory alkalosis		Peroxisomal disorders	Organic acidaemias
Hereditary fructose intolerance	Hyperammonaemia		Non-ketotic hyperglycinaemia Molybdenum cofactor deficiency	Hereditary fructose intolerance

**Table 2.3** Metabolic disorders associated with various patterns of deterioration\*.

- 4. Congenital lacticacidaemias, i.e., PDH, respiratory chain disorders, biotinidase deficiency.
- 5. Carbohydrate disorders, e.g., galactosaemia, glycogen storage disease type I hereditary fructose intolerance, fructose 1,6-bisphosphatase deficiency.

In practise, with the exception of the fatty acid oxidation defects, the majority of these disorders do not strictly present as SIDS but rather as an acute metabolic crisis with clear clinical symptoms which precedes death by hours or even a few days.

## **Reye Syndrome**

Acute hepato-encephalopathy with microvesicular fat deposition in the hepatocyte, usually during an infection, the underlying pathogenesis being acute mitochondrial dysfunction of varying aetiology. Patients can present at any age, but the condition is more common in children. Salicylates have been implicated as a trigger and their use in children is now contra-indicated, consequently, Reye syndrome resulting from salicylate use is rare. Patients present acutely with vomiting, lethargy, increased confusion often progressing to coma, seizures with decerebration and finally respiratory arrest. Hyperammonaemia, hypoglycaemia, metabolic acidosis with deranged liver function are the biochemical hallmarks. Histological findings are swollen hepatocytes, panlobular microvesicular steatosis and "typical" mitochondrial abnormalities on electron microscopy. The differential diagnosis includes a number of metabolic disorders, primarily urea cycle defects, fatty acid oxidation and ketogenesis defect, mitochondriopathies, organic acidurias, gluconeogenesis defects and fructose intolerance.

<sup>\*</sup>Adapted from Leonard and Morris<sup>39</sup>.

## **Suspected Non-accidental Injury**

False accusations of abuse can have catastrophic consequences for the family involved. It must be noted that the clinical information provided by the family and the clinicians is vital and that final diagnosis can only be made when all the facts are known. A number of metabolic disorders have come to light while infants/children were being investigated for possible abuse, but metabolic disease cannot be diagnosed if it is not suspected and if the appropriate tests are not carried out.

When assessing cases of possible abuse one should consider the following:

- 1. Do the injuries accord with the history given by the family?
- 2. Has the family suffered a similar "unexpected" death?
- 3. Is there a history of consanguinity?
- 4. Was there a prodromal illness?
- 5. Could this be due to an underlying metabolic disorder?

Glutaric aciduria type I (GA I), Menkes disease<sup>51</sup>, osteogenesis imperfecta, familial hypophosphatasia and haemophilia have all been sighted as causing damage to organs and/or tissues that have been wrongly interpreted as signs of child abuse.

GA I can lead to subdural haematoma and bilateral retinal haemorrhages very closely mimicking the shaken baby syndrome<sup>43,44</sup>. Similarly defects affecting connective tissue/bone metabolism may lead to multiple fractures and or ruptured blood vessels, which again can be misinterpreted as signs of non-accidental injury. We would therefore strongly advise that adequate material is taken in such cases to enable a full biochemical profile to be carried out and thereby exclude any underlying metabolic disorder.

## **Cardiomyopathy**

Isolated cardiomyopathy as a primary cause of death, or in conjunction with other organ involvement, is a relatively common finding for the paediatric histopathologist. The numerous causes of this condition are therefore worth expanding in some detail as correct identification of the underlying defect may allow identification and treatment of other affected family members or in some cases offer the chance for antenatal diagnosis.

#### Types of Cardiomyopathy

- 1. Dilated cardiomyopathy
- 2. Hypertrophic cardiomyopathy

#### Most common causes of metabolic cardiomyopathy

- 1. Genetic defects of sarcomeric proteins
- 2. Fatty acid oxidation defects including defects of the carnitine shuttle
- 3. Respiratory chain defects
- 4. Storage diseases
- 5. Rare disorders

## **Hypertrophic Cardiomyopathy (HCM)**

HCM is a primary disease of the myocardium with an incidence of 1:500. Distribution of hypertrophy is typically asymmetrical. Idiopathic hypertrophic subaortic stenosis (IHSS), hypertrophic obstructive cardiomyopathy, muscular subaortic stenosis, and asymmetric septal hypertrophy (ASH) are among its many synonyms. Fifty per cent of cases are familial and are inherited as an autosomal dominant trait. HCM often does not develop until after onset of puberty or adulthood. With HCM, although rare in children under 15 years of age, 6% of these children will experience sudden cardiac death.

**Histology:** cardiac muscle disorganisation (disarray), replacement fibrosis, myocyte hypertrophy, abnormalities of the small intramural coronary arteries.

Pathophysiology: diastolic dysfunction with abnormalities in ventricular relaxation.

There is a wide variability of expression within affected families. Children presenting before 4 years of age may have associated dysmorphisms consistent with genetic syndromes (i.e., Noonan Syndrome; Cardio-facio-cutaneous syndrome (CFC), Beckwith-Wiedermann syndrome), HCM with Wolff-Parkinson-White syndrome (chromosome 7q3). The disease can also be caused by a mutation in one of seven genes that encode proteins of the cardiac sarcomere. However, in practice most disease is due to mutations in 1 of 4 sarcomeric genes, defects in the other genes being reported in isolated families or individuals. These 4 genes are:

- 1. Myosin heavy chain (chromosome 14q1) at residues 403, 453, 606 and 719, associated with poor prognosis, accounts for 35% of mutations in the sarcomeric genes. Clinical features are heterogeneous with some point mutations associated with benign outcome and others with high incidence of sudden death, typically seen in young people <20 years old.
- 2. Cardiac myosin-binding protein C (chromosome 11p 11.2) this protein modulates cardiac contractility. Mutations possibly associated with late-onset hypertrophy tend to be "mild" and account for 15% of mutations.
- 3. Cardiac troponin T (chromosome 1q3) accounts for 15% of mutations.
- 4. Tropomyosin (chromosome 15q2) <5% of mutations.

## **Dilated Cardiomyopathy (DCM)**

This is the most common form of cardiomyopathy. In children under two years of age 45% of disease is due to myocarditis, 25% to endocardial fibroelastosis and 30% remains unidentified.

**Pathophysiology:** systolic dysfunction with 46% presenting with arrhythmias (mainly atrial) in children with 20–30% having familial forms, x-linked DCM being the most common form of inheritance.

- 1. Xp21: (dystrophin locus; rapidly progressive disorder in male teenagers).
- 2. Xq28: Barth syndrome: DCM; skeletal disease; neutropaenia; 3-methylglutaconic aciduria; abnormal mitochondria. Rapidly fatal in early infancy.
- 3. Mutations in the taffazin gene.
- Autosomal dominant DCM mutations in genes for actin, lamin A/C and desmin<sup>45</sup>.

## **Fatty Acid Oxidation Defects Leading to Cardiomyopathy**

Cardiomyopathy as a consequence of a fatty acid oxidation disorder can be hypertrophic or dilated. All of the following can lead to cardiomyopathy: Primary carnitine deficiency (PCD), CPTII, CAT, VLCAD, LCHAD, GAII (see section on fatty acid oxidation defects and organic acidurias).

## **Mitochondrial Respiratory Chain Disorders**

The normal heart consists of around 30% by volume of mitochondria. In many of the cardiac mitochondrial disorders, myocardial biopsy demonstrates an absolute increase in number and size of mitochondria, while the activity of mitochondrial enzymes are decreased<sup>46</sup>. Most patients have concentric hypertrophic and hypokinetic cardiomyopathy, the question of whether the dilated forms are primary or secondarily dilated is still debatable. All types of respiratory chain enzyme deficiency have been reported, but complex I deficiency is significantly more frequent<sup>47</sup>.

- 1. Mitochondrial DNA mutations mtDNA point mutations and deletions and tRNA point mutations.
- 2. Other defects of the mitochondrial respiratory chain nuclear encoded defects.

tRNA point mutations that may cause cardiomyopathy

Other novel mtDNA mutations include: cytb (15508), tRNA<sup>arg</sup> (10424), tRNA<sup>ala</sup> (5655), ND4L (10554) and ND5 (14069).

Depletion of mitochondrial DNA (60-75%) in cardiac tissue is also a reported cause of cardiomyopathy<sup>48</sup>.

Nuclear encoded defects of the respiratory chain and its assembly and organisation, e.g., histiocytoid cardiomyopathy in infancy resulting from Complex III deficiency shows normal complex activity in liver and skeletal muscle; this suggests that the defect involves a tissue-specific subunit<sup>49</sup>.

## **Storage Disorders**

Glycogen storage disease Type II – Pompe Disease. Gangliosidosis  $GM_1$ ,  $GM_2$ , MPS I, II, IV, VI, VII, Mucolipidosis II.

### **Other Rare Disorders**

- 1. Hemizygous form of Fabry disease isolated cardiomyopathy.
- 2. GSD IV mild forms may present exclusively with cardiomyopathy.
- Sengers disease defect in mitochondrial function presenting with cataracts in all cases.
- 4. Congenital defects of glycosylation (CDG).

## References

- 1. Garrod AE. Inborn errors of metabolism. Oxford: Oxford University Press, 1909.
- 2. Bennett MJ, Rinaldo P. The metabolic autopsy comes of age. Clin Chem 2001; 47(7):1145-6.
- 3. Chace DH, DiPerna JC, Mitchell BL, Sgroi B, Hofman LF, Naylor EW. Electrospray tandem mass spectrometry for acylcarnitines in dried post-mortem blood specimens collected at autopsy from infants with unexplained cause of death. Clin Chem 2001;47(7):1166–82.
- 4. Roe CR, Ding J. Mitochondrial fatty acid oxidation disorders. In: Scriver CR, Beaudet al, Sly WS, Valle D, editors. The metabolic and molecular basis of inherited disease, 8th Ed. New York: McGraw-Hill, 2000;2297–326.
- 5. Bennett MJ, Allison FA, Pollitt RJ, Variend S. Fatty acid oxidation defects as cause of sudden death in infancy. Prog Clin Biol Res 1990;321:349-64.
- Ruitenbeek W, Poelis PJ, Turnbull DM, Garavaglia B, Chalmers RA, Taylor RW, et al. Rhabdomyolysis and acute encephalopathy in late-onset medium-chain acyl-CoA dehydrogenase deficiency. J Neurol Neurosurg Psychiatry 1995;58(2):209–14.
- 7. Rinaldo P, Raymond K, Barnes CA. Medium-chain acyl-CoA dehydrogenase deficiency: Sudden and unexpected death of a 45-year-old woman. J Inher Metab Dis 1999;22:104.
- 8. Gregersen N, Blakemore A, Winter V, Andresen BS, Kolvraa S, Bolund L, et al. Specific diagnosis of medium-chain acyl-CoA dehydrogenase (MCAD) deficiency in dried blood spots by a polymerase chain reaction (PCR) assay detecting a point mutation (G985) in the MCAD gene. Clin Chim Acta 1991;203:23–34.
- 9. Brivet M, Boutron A, Slama A, Costa C, Thuillier L, Demaugre F, et al. Defects in activation and transport of fatty acids. J Inher Metab Dis 1999; 22:428–41.
- 10. Wanders RJA, Vreken P, Den Boer MEJ, Wijburg FA, Van Gennip AH, Ijlst L. Disorders of mitochondrial fatty acid acyl-CoA (-oxidation. J Inher Metab Dis 1999;22:442–87.
- 11. Chalmers RA, Stanley CA, English N, Wigglesworth JS. Mitochondrial carnitine acylcarnitine translocase deficiency presenting as sudden neonatal death. J Pediatr 1997; 131:220-5.
- 12. Olpin SE, Allen JC, Bonham JR, Clark S, Clayton PT, Calvin J, et al. Features of CPTI deficiency. J Inher Metab Dis 2001;24:35–42.
- Pollitt RJ, Olpin SE, Bonham JR, Cahalane SF, Naughten E. Late-presenting carnitine transport defect. Enzyme and Protein 1993;3:175.
- De Vivo DC, Tein I. Primary and secondary disorders of carnitine metabolism. Internat Paediatr 1990;5:134-41.
- Manning NJ, Olpin SE, Pollitt RJ, Webley J. A comparison of [9,10-3H]myristic and [9,10-3H]palmitic acids for the detection of fatty acid oxidation in intact cultured fibroblasts. J Inher Metab Dis 1990;13:58-68.
- 16. Olpin SE, Manning NJ, Pollitt RJ, Bonham JR, Downing M, Clark S. The use of [9,10-3H]myristate, [9,10-3H]palmitate and [9,10-3H]oleate for the detection and diagnosis of medium- and long-chain fatty acid oxidation disorders in intact cultured fibroblasts. In: Quant PA, Eaton S, editors. Current views of fatty acid oxidation and ketogenesis (Proceedings of the 4th International Symposium on Fatty Acid Oxidation) New York: Plenum Press. Adv Exp Med Biol 1999;466:321-5.
- 17. Nada MA, Rhead WJ, Sprecher H, Schulz H, Roe CR Evidence for intermediate channelling in mitochondrial beta-oxidation. J Biol Chem 1995;270:530–5.
- 18. Roe CR, Roe DS. Recent developments in the investigation of inherited metabolic disorders using cultured human cells. Mol Genet Metab 1999;68(2):243–57.
- 19. Ijlst L, Ruiter JP, Hoovers JM, Jakobs ME, Wanders RJ. Common mis-sense mutation G1528C in long-chain 3-hydroxyacyl-CoA dehydrogenase deficiency. Characterisation and expression of the mutant protein, mutation analysis on genomic DNA and chromosomal localization of the mitochondrial trifunctional protein alpha subunit gene. J Clin Invest 1996;98:1028–33.
- 20. Munnich A, Rotig A, Cormier-Daire V, Rustin P. Clinical presentation of respiratory chain deficiency. In: Scriver CR, Beaudet al, Sly WS, Valle D, editors. The metabolic and molecular basis of inherited disease, 8th ed. New York: McGraw-Hill, 2000;2261–74.
- Soussi B, Idstrom J, Schersten T, Bylund-Fellenius A. Cytochrome c oxidase and cardiolipin alterations in response to skeletal muscle ischaemia and reperfusion. Acta Physiol Scand 1990;138:481–7.
- 22. Pitkanen S, Feigenbaum A, Laframboise R, Robinson BH. NADH-coenzyme Q reductase (complex I) deficiency: heterogeneity in phenotype and biochemical findings. J Inher Metab Dis 1996;19:675–86.

- 23. Robinson BH, Ward J, Goodyer P, Baudet A. Respiratory chain defects in the mitochondria of cultured fibroblasts from three patients with lacticacidaemia. J Clin Invest 1986;77:1422-7.
- 24. Robinson BH, De Meirlier L, Glerum P, Sherwood G, Becker L. Clinical presentation of mitochondrial respiratory chain defects in NADH-coenzyme Q reductase and cytochrome oxidase. Clues to pathogenesis of Leigh disease. J Pediatr 1987;110:216-22.
- 25. Robinson BH, Glerum DM, Chow W, Petrova-Benedict R, Lightowlers R, Capaldi R. The use of skin fibroblast cultures in the detection of respiratory chain defects in patients with lactic acidaemia. Pediatr Res 1990;28:549–55.
- 26. Van Erven PM, Ruiten W, Gabreels FM, Renier WO. Mitochondrial encephalopathy: association with an NADH dehydrogenase deficiency. Arch Neurol 1987;44:775–8.
- 27. Robinson BH, Oei J, Sherwood WG, Applegarth D, Wong L, Haworth J, et al.. The molecular basis for the two different clinical presentations of classical pyruvate carboxylase deficiency. Am J Hum Genet 1984;36:283–94.
- 28. Wanders RJA, Ruiter JPN, Wijburg GA. Studies on mitochondrial oxidative phosphorylation in permeabilised skin fibroblasts: application to mitochondrial encephalomyopathies. Biochim Biophys Acta 1993;181:219–22.
- 29. Fischer JC, Ruitenbeek W, Stadhouders AM, Trijbels JM, Sengers RC, Janssen AJ, et al. Investigations of mitochondrial metabolism in small human skeletal muscle biopsy specimens. Improvement of preparation procedure. Clin Chim Acta 1985;145:89–94.
- 30. Glerum M, Yanamura W, Capaldi R, Robinson BH. Characterisation of cytochrome oxidase mutations in human fibroblasts. FEBS Lett 1988;236:100-104.
- Moreadith RW, Batshaw ML, Ohinishi T, Kerr D, Knox B, Jackson D, et al. Deficiency of ironsulphur clusters of mitochondrial reduced nicotinamide-adenine dineucleotide-ubiquinone oxidoreductase (complex I) in an infant with congenital lactic acidosis. J Clin Invest 1984;74:685–97.
- 32. Rustin P, Chretien D, Bourgeron T, Gerard B, Rotig A, Saudubray JM, et al. Biochemical and molecular investigations in respiratory chain deficiencies. Clin Chim Acta 1994;228:35–51.
- 33. Besley GT, Wraith JE. Lysosomal disorders Mini-symposium: Metabolic disease. Curr Paediatr 1997;7:128-34.
- Van Maldergem L, Jauniaux E, Fourneau C, Gillerot Y. Genetic causes of hydrops fetalis. Paediatrics 1992;89:81-6.
- 35. Poggi-Travert F, Fournier B, Poll-The BT, Saudubray J-M. Clinical approach to inherited peroxisomal disorders. In: Roels F, De Bie S, Schutgens RB, Besley GT, Eds. Diagnosis of human peroxisomal disorders a handbook. J Inher Metab Dis 1995;18:1–18.
- 36. Espeel M, Van Limbergen G. Immunocytochemical localisation of peroxisomal proteins in human liver and kidney. J Inher Metab Dis 1995;18:135–54.
- 37. Kerckaert I, De Craemer D, Van Limbergen G. Practical guide to morphometry of human peroxisomes on electron micrographs. J Inher Metab Dis 1995;18:172–80.
- 38. Olpin SE, Manning NJ, Pollitt RJ, Clarke S. [9,10-3H]Oleic acid for the improved detection of long-chain fatty acid oxidation defects in intact cells. J Inher Metab Dis 1997;20: 415–19.
- 39. Leonard JV, Morris AAM. Inborn errors of metabolism around the time of birth. Lancet 2000;356:583-7
- Beckwith JB. Observations on the pathological anatomy of the sudden infant death syndrome.
   In: Bergman AB, Beckwith JB, Ray CG, editors. Sudden Infant Death Syndrome. Seattle: University of Washington Press, 1970:83-102.
- 41. Sinclair-Smith C, Dinsdale F, Emery J. Evidence of duration and type of illness in children found unexpectedly dead. Arch Dis Child 1976;51:424–8.
- 42. Saudubray J-M, Charpentier C. Clinical Phenotypes: Diagnosis/Algorithms. In: Scriver CR, Beaudet al, Sly WS, Valle D, editors. The metabolic and molecular basis of inherited disease, 8th ed. New York: McGraw-Hill, 2000;1327–403.
- 43. Hoffman GF, Athanassopoulos S, Burlina AB, et al. Clinical course early diagnosis, treatment, and prevention of disease in glutaryl-CoA dehydrogenase deficiency. Neuropaediatrics 1996;27:115–23.
- 44. Wilkins B. Head injury abuse or accident? Arch Dis Child 1997;76:393-7.
- 45. Tsubata S, Bowles KR, Vatta M, Zintz C, Titus J, Muhonen L. Mutations in human delta-sarco-glycan gene in familial and sporadic dilated cardiomyopathy. J Clin Invest 2000;106(5):655–62.
- 46. Peters TJ, Wells G, Oakley CM, Brooksby IA, Jenkins BS, Webb-Peploe MM, et al. Enzymatic analysis of endomyocardial biopsy specimens from patients with cardiomyopathies. Br Heart J 1997;39:1333.
- 47. von Kleist-Retzow JC, Cormier-Daire V, de Lonlay P, Parfait B, Chretien D, Rustin P. A high rate of parental consanguinity (20–30%) in cytochrome oxidase deficiency. Am J Hum Genet 1998;63:428.

- 48. Marin-Garcia J, Ananthakrishnan R, Goldenthal MJ, Pierpoint ME. Biochemical and molecular basis for mitochondrial cardiomyopathy in neonates and children. J Inher Metab Dis 2000;23: 625–33.
- 49. Gibert-Barness E. Cardiovascular system, Part II. In: Gibert-Barness E, editor, Potter's pathology of the fetus and infant. St. Louis: Mosby Year Book, 1997.
- 50. Lamhonwah A-M, Olpin SE, Pollitt RJ, Vianey-Saban C, Divry P, Guffon N, Besley GTN, Onizuka R, De Meirleir LJ, Cvitanovic-Sojat L, Baric I, Dionisi-Vici C, Fumic K, Maradin M, Tein I. Lack of Genotype-phenotype correlation in eleven individuals with novel OCTN2 mutations: Early carnitine therapy prevents cardiomyopathy. Am J Med Genet 2002;111(3):271–84.
- 51. Adams PC, Strand RD, Bresnan MJ, Lucky AW. Kinky hair syndrome: serial study of radiological findings with emphasis on the similarity to the battered child syndrome. Paediatr Radiol 1974;56:117-18.
- 52. Okun JG, Kolker S, Schulze A, Kohlmuller D, Olgemoller K, Linder M, Hoffmann GF, Wanders RJA, Mayatepek E. A method for quantitative acylcarnitine profiling in human skin fibroblasts using unlabelled palmitic acid: diagnosis of fatty acid oxidation disorders and differentiation between biochemical phenotypes of MCAD deficiency. Biochim Acta 2002;1584:91–8.

## 3. The Disputed Death

C. Gray

If I should die and leave you here awhile, Be not like others, sore undone, who keep Long vigils by the silent dust, and weep. For my sake – turn again to life and smile, Nerving thy heart and trembling hand to do Something to comfort other hearts than thine. Complete those dear unfinished tasks of mine And I, perchance, may therein comfort you.

[Anonymous – variously attributed]

O death, where is thy sting-a-ling-a-ling,
O grave, thy victory?
The bells of Hell go ting-a-ling-a-ling
For you but not for me.

[British soldiers during the Great War]

Rational people wish to die in their own bed, in the bosom of their devoted family, of a short and painless but necessarily severe illness supervised by a kindly and efficient physician who accurately certifies the cause of death to the satisfaction of all concerned.

Alas, this does not happen in an important minority of cases and there is nothing worse than dying for the wrong reason. Disputes touching upon a death are difficult for all those concerned, especially the relatives who have to mount an argument at a time of grief. Problems around a death become one of the most intense interfaces between ordinary folk and the learned professions and state authorities. Disputed deaths consume substantial resources of time and money in public organisations and yet form a substantial stream of remunerated work for lawyers and related professionals.

Disputes on a death can have widespread repercussions in history, public life, medicine, the law and not least upon the family of the dead person. The peculiar death of a predecessor in the family can have effects decades later and may blight the lives of younger family members. The media have an intense interest in sensationalising death: morbid curiosity combines with selling newspapers and filling air time in exaggerating unusual aspects of deaths and their disputes.

Disputed death is a huge subject. In this chapter I shall introduce and examine the scope of disputed death from the viewpoint of the investigating pathologist and related professionals, emphasising always the consumers' point of view and the societal framework. Each section alone could be the subject of a detailed academic review for which we do not have space. I intend merely to explore and illustrate the range of disputes which arise following death and to show where pathologists' efforts should be directed.

## **Popular Culture**

Disputed deaths are the staple basis of crime fiction: the detective story, the murder mystery, the house party game. Many television hours are devoted to the Inspector (Morse, Wexford, Frost, Japp), the elegant amateur sleuth (Miss Marple, Hercules Poirot, Wimsey) and their professional helpers (Quincey, Ryan) in unravelling the plot to explain how Miss Scarlett came to be lying in the Library fatally injured by the Candlestick.

## **Urban Legends**

Myths and urban legends abound in conversation and sensational gossip. Obituary columns are the best-read sections of newspapers and especially medical journals. Sudden and problematic deaths are the delight of the dinner table, the glee of the bowling club and, "I know a feller, who knows a bloke who lost the body of his aunt on the channel ferry. It seems she was rolled in a roll of carpet . . ." Black humour is everywhere in Britain and at the root of it lies death in all its peculiar forms. Few can resist the intrinsic humour of the man dying in the act of adulterous sexual intercourse. The story is improved if he had a dangerous occupation, banged his head earlier in the day, had a suicide note in his pocket, had intoxicants present in his body, had complained to police a few days earlier of receiving death threats, was doing it while driving his car or was caught on closed circuit TV.

### **Celebrated Cases**

The deaths of some individuals have become embedded in the popular history of the age. The suicide of Hitler in 1945, the death of Marylin Monroe in 1962, the assassination of President Kennedy in 1963, the death of Rudolf Hess in 1987, the finding in the ocean of the body of Robert Maxwell who was missing from his yacht, the death of Diana, Princess of Wales in 1998, remain controversial in many aspects. Conspiracy theorists endlessly discuss inadequate evidence and reach alarming conclusions. Did Hitler escape alive? Where are his remains? Did Monroe commit suicide or was it murder? How many bullets struck Kennedy? How many assassins fired shots? Was there a conspiracy to kill the President? Was the dead prisoner in Spandau Prison really Rudolf Hess, former Deputy Führer of the Third Reich?\frac{1}{2}. Was Maxwell's death an accident or suicide or even homicide? Was Maxwell Israel's spy?\frac{2}{2}. How did Diana come to be involved in the car crash? Could different medical help have saved her life? Sudden death of celebrities can become problematic if investigation is inadequate or the evidence incomplete or concealed.

## **Bases of Dispute**

The common bases of disputes concerning death are given in Table 3.1. Simplistically, a death is disputed if someone is making an argument about it. So, I would define a disputed death as a death in which interested parties remain in conflict over its facts, causation, liabilities or consequences.

The scope of disputes in deaths is wide and this section introduces examples of the common classes of dispute.

#### **Fact of Death**

The question whether someone is dead or alive arises in clinical practice and over missing persons. The diagnosis of death in the unconscious brain-damaged patient has some technical problems. Although well-established in Western medical practice in the context of organ transplantation from "brain dead" donors, the concept and diagnosis of brain death remains controversial around the world, especially in Japan. Readers are referred to Margaret Lock's excellent examination of the topic<sup>3,4</sup>.

The classic examples of missing persons dead or alive are Martin Bormann and Lord Lucan. Bormann was a member of the Nazi elite who disappeared after the storming of Berlin by the Russian Army in 1945. Did he escape from Berlin or die

Table 3.1 The disputed death.

Bases of dispute	Parties to a death			
Fact of death	Relatives			
Identity of remains	State			
Cause of death	Defendants			
Time of death	Political factions			
Accident, suicide or homicide	Police			
Criminal liability and conspiracy	Hospital			
Civil liability and negligence	Doctors/nurses			
Use of human tissues	Employer			
Disposal of the dead	Military			
Unresolved grief and emotion	Insurers			
Public policy	Parties with a duty of care			
Lawyers/professionals				
Common disputed death scenarios	Common causes of death in homicide			
Accident, suicide or homicide	Beating			
Infant death	Stabbing			
Industrial disease	Head injury			
Hospital mishap	Asphyxia			
Deaths in custody	Gunshot wounds			
Major disasters	Poisoning			
Genocide/assassination	Collision with a vehicle			
Organ retention	Burning			
Missing person/body found	Drowning			
Death overseas	Throwing from a height			
No body	Multiple assaults			
VIPs/historical figures				

in the fighting?<sup>5</sup> If he survived he would now be 101 years old! Lucan was a minor British aristocrat and gambler who disappeared after the murder of a domestic servant. Is he dead or alive somewhere? TV programmes have covered the conflicting evidence of these and similar cases.

Over the years many distinguished persons from Amy Johnson to Glenn Miller have gone missing when their aircraft failed to arrive. These are missing and presumed dead, but sometimes the wreck of the aircraft and human remains are found many years later<sup>6</sup>.

The water speed adventurer Donald Campbell was presumed dead after the wreck of his craft *Bluebird II* on Coniston Water in 1962. Remarkably, only in 2001 were the remains of the craft and the missing man finally found after dives by enthusiasts.

These are examples of literally disputed deaths but pathologists have no role until the suspect person is found.

### **Identification of Human Remains**

The identification of human remains can be problematic. Identification depends upon comparison of identifying physical features – such as DNA, fingerprints, dental state – in the remains with the known features of the possible missing person or persons. Sufficient points of identity must be found to confirm the match and there must be no unexplainable discordant evidence. Occasionally, putative identifications are disputed; usually in the circumstances of incomplete or degraded human remains and incomplete or missing personal records. One body found after the Kings Cross Underground Station fire remained unidentified and was buried as an unknown man. Stowaways hiding in the undercarriage of airliners commonly die en route of cold and hypoxia and fall to earth as the aircraft approaches the UK airport. Although the bodies are well preserved, the complete absence of missing person information from the country of origin precludes identification.

### **Cause and Time of Death**

Cause and time of death are common problems in death investigation. The fundamental facts to be determined by an inquest in England and Wales touching upon a death are: who it was, when it was, where it was and by what means. Many disputes lie in this territory. Examples of these are explored below.

## Accident, Suicide or Homicide?

Some deaths could variously be the result of accident or suicide or homicide. A farmer found shot in a field with his shotgun beside him; an elderly woman found drowned in a canal; a drunken student falling from a bridge: these are examples in which the three traditional possibilities are credible and must be addressed. In homicide cases, the accused person may deny the accusation and dispute his part in the death. The Defence approach to be privide in a proposal below. In page

In homicide cases, the accused person may deny the accusation and dispute his part in the death. The Defence approach to homicide is examined below. In more grandiose cases, a death may allegedly be part of a wider conspiracy by political opponents, police, militia or warring states. Several large investigations into alleged genocide are in progress around the world.

## Negligence

In circumstances where a duty of care is owed – a patient in hospital, the employee in the industrial plant, the airline or railway passenger, the diner in a restaurant – negligence may cause or contribute to the death. Many cases in civil law are pursued in breach of duty in the settings of medical mishap, transport accident, industrial disease, accidents and deficient services.

## Organ Retention and Disposal

Further disputes arise in the odd circumstances of body parts and organs retained by pathologists or institutions, and unusual disputes about disposal of the dead. The man who wishes to bury the body of his wife in the garden, the pathologist who retains an interesting specimen without permission, the sea dog whose family wishes to bury him at sea although he died in Birmingham: these are examples of disputes in this area.

## **Relatives and Public Policy**

Relatives can remain disputatious for years for differing reasons; these may or may not have sound bases. The emotional needs of relatives are discussed below. Finally, disputed deaths can feed into changes in public policy. Typically, road crashes show the need for improvements to roads and signals in the locality and vehicle design. Mass transport disasters bring calls for improved safety standards and operational management. And recurrent problems in hospitals, police stations and organisations can eventually identify strategies for prevention of medical errors and deaths in custody and for improving the care of military recruits, prisoners, students and other inmates of institutions.

## **Parties to a Disputed Death**

Parties to a death are various and may include some of those shown in Table 3.1. Most deceased persons have relatives, and disputes after death usually involve the next-of-kin or other relatives on behalf of the interests of the dead person against some other party. Some deceased persons have no known relatives and their interests must be represented by the official solicitor or the local authority in some circumstances. An unidentified person may have been murdered – such is the position of many of the victims of genocide found in mass graves – and yet accused persons can be tried for the murder or genocide.

### The State

The state is involved in the administration following a death in many ways. A death must in the UK by law be registered with the Registrar of Births, Marriages and Deaths. The death may fall under the jurisdiction of Her Majesty's Coroner (England and Wales, Northern Ireland) or the Procurator Fiscal (Scotland). Diverse state agencies may become involved, such as the Health and Safety Executive in the case of an industrial accident or a transport disaster. Hospitals, police, social and emergency services and local authorities may be criticised for alleged failings in

service or quality of care. In the worst disputes over mass disasters or child abuse – or any event of significant public interest – a public enquiry may be held, chaired by a judge or senior barrister.

#### **Defendant**

The defendant in a criminal case has an obvious interest in homicide and defendants in civil actions may also contest liability in negligence actions. A person's employer has duties of care and may be criticised following a death in the workplace. Insurers are obliged to pay out life policies on death but may contest some deaths. The insured person may have acted with contributory negligence or lack of care and insurers may not pay out for suicide within some years of the start of a policy.

### **Lawyers and Professionals**

Lawyers, of course, find substantial professional work in providing legal services after a death, from probate and contested wills, to criminal and civil representation. Medical and other professional experts also find remunerative work in this field.

### **Needs of Relatives**

In most disputed deaths, the relatives are the aggrieved party and much of the following discussion relates to investigating a disputed death and dealing with the relatives.

### **Relatives' Emotions**

Relatives' emotions are complex and – particularly after a sudden, untimely, violent or problematic death – will include the grief reaction, anger, blame and periods of incapacity. For many relatives, the problematic death of their loved one presents them with the worst trial of their lives at a time when they are necessarily impaired in dealing with it.

Modern culture regards death as distasteful and a manifestation of failure. The decline of traditional religion and the rarity of deaths in childhood mean that many people have little experience of dealing with death in the family until late adulthood. Loss of spiritual faith concentrates the feelings of the bereaved upon the physical remains of the deceased and the locus of death. In the UK, roadsides show many decaying floral shrines placed exactly at the place of the fatal crash. Relatives have little experience of the language and practice of pathologists, coroners and lawyers and may have distorted expectations of the official processes from the media and television drama.

Recent public outrage at the Alder Hey organ retention affair shows that professional customs have become separated from public perception and sentiment. Relatives' contact with professionals becomes highly focussed and amplifies the content of discussions. What to the experienced professional may be a routine detail; to the bereaved relative can become a nightmare.

Health care professionals can themselves be shocked at their own emotional reactions when dealing with the sudden illness and death of their loved ones. Doctors and nurses commonly admit: it's different when it's you.

Relatives in dispute may simply be seeking explanations of questions of fact. They may also have misunderstandings owing to lack of information and experience in the field. This is especially true following hospital mishaps. Early contact with senior doctors and nurses can explain what happened and why it happened; this can allay concerns and lead to acceptance of the outcome without pursuing a legal claim.

Other relatives may be determined to attribute blame on responsible individuals or organisations and may pursue legal proceedings beyond their likelihood of success. Compensation neurosis can feature in the rest of their lives. Others form pressure groups, determined to reform the law to prevent similar failings or to pursue to "justice" an individual professional.

## **Dealing with Relatives**

All people are different. But, usually, relatives are in need of information by explanation and support. This can be delivered by their general practitioner, hospital doctors, nurses and the coroner's officer or the pathologist. Some will rest content at this point. Others will seek retribution and blame, or restitution and money and for this they will need to consult a solicitor. Solicitors will then need expert opinion from independent practitioners in the relevant field.

Pathologists in some centres have formalised arrangements to meet relatives' needs: in Scotland there are "Death Clinics" and in England there are examples of regular post-mortem clinics. Many pathologists will meet relatives and their general practitioner on request to discuss the death of a patient. Pathologists meeting relatives should start with a firm expression of sympathy and condolence. They should then explain their role and the limitations of the evidence available. The relatives' questions and problems should be explored. The routes of investigation available should be explained and they should be put in touch with appropriate agencies. Relatives have difficulty taking in what is said and may become distressed at some point in an interview. It is helpful to provide written explanations in the form of leaflets and reports. A written explanation of the points raised sent some days later may be helpful.

Following a tragic hospital mishap, perhaps the wrong drug administered or death under anaesthesia, the reactions of the bereaved family member typically follow a pattern. Initially there is shock, disbelief and distress. This turns to anger and distress, peaking around the time of the funeral – typically about a week or ten days in the UK – which then declines. After several months, distress is reduced but remaining questions are pursued. Distress typically returns on the anniversaries of the death and grief can remain unresolved for many years owing to uncertainty, guilt and resentment. Case-hardened health care professionals can fail to appreciate the emotional shock and intellectual incapacity caused in close relatives by the sudden unexpected illness and death of a family member.

The National Health Service (NHS) "help lines" established after the organ retention affair in the UK in 2001 illustrated well the circumstances of unresolved grief. Pathologists faced many relatives who remained angry and distressed decades after a death. Cases in Harrogate included a baby who died in infancy in 1950, guilt over the taking of a drug in pregnancy in 1981, a lost grave, suspicion of malpractice, a baby "taken away by the doctor in his car" and women who had very early miscarriages but were told they had "lost the baby". Common contributory factors in these cases were the failure of information given by professionals at the time and perhaps misunderstanding by the bereaved person themselves. This allowed

unanswered questions to remain. In some cases, distress was relieved by straightforward explanation of what happened and why. The response to the help-line enquiry was the first explanation the caller had had from the health care professions since the death many years earlier.

## **Common Disputed Death Scenarios**

Regular problems in disputed death are shown in Table 3.1. Forensic pathologists commonly deal with homicides, suicides and accidents, and detailed analyses of these cases can be found in standard works and elsewhere in this and the previous volume of *Essentials of Autopsy Practice*<sup>7</sup>.

#### Homicide

The defence approach to homicide is discussed below. Apart from the interest of the defendant, relatives of victims and the general public remain intensely interested in murders and murderers, as shown by the press coverage of the cases of Shipman, Hindley and Hanratty; the large space devoted to "true crime" descriptive works in bookshops; and the immense popularity of fiction and drama in the "crime" genre.

### Suicide

In England and Wales, an inquest verdict of suicide requires clear evidence that the deceased intended to kill himself<sup>8</sup>. While the cause of death may be reliably determined to be some form of self-destructive act – commonly gassing by car exhaust fumes, hanging, drug overdose or shooting – conclusive evidence of intent to die is often lacking. Sometimes the deceased was in a personal crisis at the time – usually financial or relationship-related – with consequential emotional disturbance or mental illness. Suicide notes can be ambiguous, and intoxication with alcohol is commonly present in the suicide. Contributory factors in suicide include: depressive illness, chronic physical illness and pain, bereavement, unemployment and financial problems, addictions and marital dysfunction. But many suicides come "out-of-the-blue" with no premonitory events and as complete and horrific surprises to the next-of-kin. The highest suicide risks are men in their forties and young male prisoners in their first fortnight of incarceration. Apparent suicides can turn out to be concealed homicides.

### **Accidental Death**

Accidental death needs to be distinguished from homicide and suicide. But many accidents become subject to dispute including questions of causation and liability in car crashes, accidents at work, falls down stairs and inevitable accidents sustained during do-it-yourself handyman activities.

The recovery of an immersed body starts an investigation as drowning may be accident, suicide or homicide. Not all immersed bodies are actually drowned.

Deaths in the setting of autoerotic sexual asphyxia – also known as "lethal paraphilia" – are recognised usually to be accidental but may be mistaken for suicidal hanging. The sexual nature of the scene may lead embarrassed relatives to rearrange the body and conceal evidence before calling the police.

#### **Road Crashes**

Road crashes are perhaps the commonest disputed deaths, with many thousands occurring in the UK each year. The parties involved include drivers, passengers, occupants of other vehicles, pedestrians and people nearby in the environment: any of these can be killed. Police forces have departments skilled in the analysis of crashes including examination of the vehicles involved, the road surfaces and markings, and the driving conditions. Pathologists are expected to describe and interpret the patterns of injury sustained, which can be distinctive and informative. Occasionally, forensic scientists may be asked to identify human traces on suspect vehicles in "hit and run" crashes. Toxicologists are asked to detect any alcohol or drugs causing intoxication in drivers and pedestrians. The local authority is responsible for the road surface, alignment and signage. Vehicle manufacturers are responsible for the safety of their vehicles as sold and their owners for their maintenance.

Causes of road crashes are various: usual causes include driver error, vehicle failure, road conditions, intoxication and natural illness. Single-vehicle accidents offer a conundrum: has the driver collapsed from natural illness, made a driving error, been intoxicated or driven deliberately off the road? At least some single-vehicle accidents are concealed suicides: I have known one case where a grief-stricken driver returned deliberately to crash his car at the exact spot where he had caused the death of his girlfriend years earlier in a road crash.

Crash drivers who survive may be subject to criminal proceedings for various serious motoring offences.

Some crash victims have suffered homicide by malicious running over. Also, placing the body in the road to sustain confounding post-mortem injuries is sometimes seen after homicide.

Road traffic incidents represent a rare field of trauma in which scientific study has been extensive, using the familiar manikins or "crash dummies" combined with engineering devices to measure force. Unfortunately, real crashes have erratic effects: any injury can result from any type of collision at any speed. There have been fatal, low-speed impacts and freak survival from high-speed crashes. One passenger may be killed while another can "walk away" unhurt.

### **Infant Death**

There is nothing sadder than a dead baby and all infant deaths are problematic and may be disputed. Nowadays, infant deaths from natural causes are fewer so those which occur become more suspicious. Sudden infant death with no evidence of injury may remain unexplained and parents may have difficulty in accepting the tragic event. Some infants presenting as sudden infant death may actually be homicide, and pulmonary haemorrhage may raise suspicion of asphyxia by smothering<sup>9</sup>. Controversy has raged over the likely incidence of homicide presenting as sudden infant death syndrome. Charitable interests representing bereaved parents have objected to the aspersions cast by experienced forensic pathologists. Famously, Professor Green urged pathologists to "think dirty" A classical difficulty is the dead infant with signs of abuse found in company with both parents, both of whom deny responsibility.

#### **Industrial Disease**

Industrial diseases are prescribed under various acts and necessitate an inquest in England and Wales. Relatives may have difficulty assembling the evidence needed to pursue their claim after death. Pathological evidence should be preserved but difficulties with organ retention mean that fewer organs such as lungs are now being retained for further study. Asbestos is the classical example for post-mortem dispute. The injurious exposure is typically decades in the past from when records of employment and exposure are lacking – indeed, the employing company may no longer exist. A civil claim can be mounted for malignant mesothelioma or carcinoma of lung if asbestos fibre counts in lung tissue, histology of the neoplasm and evidence of occupational or environmental exposure can be established and documented.

Persistent disputes include cases of exposure of military personnel to deliberate hazards – atomic bomb tests, nerve gases, biological warfare agents – and the former forces involved in the Gulf War, some of whom have become very ill and some have died – the so-called "Gulf War Syndrome".

## **Hospital Mishaps**

Hospital mishaps are perhaps the commonest disputed deaths. Many relatives turn out to have been misinformed about, or to have an unrealistic expectation of, the patient's prognosis even in an elderly person who was seriously ill. Junior nurses and non-clinical staff may have been routinely optimistic and inappropriately reassuring. It can be difficult to identify the grade of nurse one is speaking with. Relatives are shocked by the death when they were expecting recovery. They then are told or – even worse – find out themselves that something important has gone wrong in the care of the patient. Doctors and nurses alike can be very bad at explaining matters and relatives then become angry at perceived "cover-up" or denial by hospital authorities. Health care professionals commonly underestimate the need of relatives for constant explanations of what is going on with the patient.

A hospital mishap can lead to a large inquest with legal representation of all parties, hospital doctors, nurses and managers squirming in the witness box, and angry relatives denouncing inferior quality of care. Unfortunate or embarrassing entries in hospital case records may be read out in court in full, and important notes of the patient's condition at material times can be lacking. The pathologist's duty is to explain the medical cause of death, and to interpret the importance of the "mishap" in the sequence of events. Coroners should obtain the services of an "expert assessor" - a doctor expert in the field from another region, perhaps nominated by a professional society or a Royal College - to evaluate the questions of deficiency in clinical practice. Few pathologists have direct personal experience of modern clinical practice in other medical specialities and pathologists must avoid substituting their own "amateur" judgement for that of a clinical expert in the field. Many hospital mishaps occur in patients already seriously ill from natural conditions. The burden of natural disease and the expectation of life should always be evaluated and explained to the coroner's court.

## **Deaths in Custody**

Deaths in custody are exquisitely problematic and represent some of the most disputed deaths. *Copwatcher* – a pressure group suspicious of the police – have a web site devoted to the subject<sup>14</sup>. The case of Christopher Alder in Hull went to an inquest in 2000, which lasted over six weeks in the coroner's court, after which the jury gave a verdict of "unlawful killing". This was confirmed by judicial review. A trial in the Crown Court of five police officers for "Wilful neglect amounting to misconduct in a public office" ended in acquittal. Humberside Police later reviewed training in relation to care and custody of persons detained and provided extensive first-aid training for its officers<sup>11</sup>.

Of course, no-one should die in custody but custody is defined widely to include all prisoners in penal institutions and all persons under arrest or being pursued by police, wherever the death occurs, even in hospital. Serving prisoners in HM prisons include many elderly and ill people. Inevitably, some will collapse and die of natural conditions such as heart disease, just as occurs in the wider community. Suicide of prisoners is a persistent problem. Young men within weeks of their first admission to prison are at most risk but medical admission assessments are not good at identifying those prisoners at greatest risk. Care by prison staff is also not good at preventing suicidal attempts and prisoners can be ingenious in achieving self destruction within limited physical means. Mental illness, drug addiction, substance abuse and trafficking and violence are endemic in prison populations and are very difficult to manage.

The death in police custody is the most problematic. Most such cases involve a victim who is mentally ill, intoxicated by drugs and/or alcohol and in a physiologically disturbed state, termed excited delirium in the USA or exhaustional mania in the UK, in which adrenalin and related systems are overactive. Death may follow a struggle between the prisoner and officers and involve physical restraint and the risk of "restraint asphyxia". In other cases, police fail to recognise that the prisoner is dangerously ill or unconscious without protected airways. Typically, the prisoner is under arrest for aggressive behaviour and may be obviously intoxicated by alcohol or drugs, particularly cocaine or amphetamines. Signs of head injury, hypoglycaemia and decreasing conscious level from overdose can be mistaken for drunkenness or aggressive behaviour and the prisoner may be left to "sleep it off". The prisoner is then found either dead or seriously ill and dying on a later check. Police have statutory duties of care to those in custody and must practise within strict rules<sup>12</sup>. Failure to follow their own rules can lead to serious disputes about responsibility for a death. No unconscious person should remain in a police station and better training of police officers and staff in resuscitation are desirable. Inevitably, many prisoners present police with severe challenges in safe custody: police stations on weekend evenings usually fill with aggressive and intoxicated clients under arrest for various offences. It is difficult for police to identify clients at risk of death while trying to carry out their duties.

Overseas, the standards may be different and some states are alleged to maintain instruments of torture in police stations. Allegations of police and military brutality are frequent in areas of conflict around the world. *Amnesty International* has huge records of abuse and torture in many nations around the world, including Europe.

## **Major Disasters**

Major disasters with multiple fatalities raise many disputes. Police, HM coroner and pathologists have the task of determining identity and causes of death. Failures of technology or human management may have contributed to an aviation or railway disaster. Issues may be addressed months or years after the events by a public enquiry. Criminal liability may be followed up decades later, as happened in the Lockerbie air disaster case in which an airliner was destroyed by a bomb over Scotland and accused persons were eventually brought to trial at the Scottish High Court sitting in the Netherlands after a complex diplomatic negotiation<sup>13</sup>. The insurance claims following an accident or disaster can be very complex.

#### Genocide

Massacres of populations have occurred around the world: leading examples are Rwanda, the Balkan war and Indonesia. Pathologists and anthropologists may work on behalf of international organisations, such as the United Nations, in exhuming and examining the remains of people found in mass graves. The difficulties are severe: bodies may have been years in the ground and reduced by decomposition; evidence of identification can be missing and crime scenes may have been subject to tampering and disturbance. Investigating personnel may be hazarded by attacks by irregular forces or by booby-trap devices such as land mines and grenades.

The International Criminal Tribunal for the former Yugoslavia - which sits at The Hague in The Netherlands – is an example of an international court with jurisdiction over war crimes. Its focus is on gathering evidence to convict those in control or responsible for the policy: the political and military leaders who directed the systematic deportation or destruction of a population or ethnic group for political, religious or sectarian motives. The crime of genocide is committed in the minds and directions of state leaders and military officers who form and carry out the plans rather than the individual acts of violence committed by individual soldiers. War crimes have special difficulties in their prosecution. It is difficult to identify which soldier did what to whom; whether he was following orders and if so who gave his orders. Generals typically claim that the crimes which later come to light were not their intention but were the result of excessive zeal by junior ranks. They may further claim that their country was at war or in an armed conflict and that their forces were fighting in defence of their motherland against insurgents or terrorists. Victims of massacres may be described as terrorists or the "enemy within" despite being the indigenous population of men of all ages. Trials for war crimes are perhaps the most elevated tribunals for determining disputed deaths.

Past terrorists can become today's statesmen if they renounce violence. Many leaders of former colonies can proudly claim to have been jailed by the British. Political developments can lead to re-appraisal of terrorist atrocities or conflicts decades later as shown by the "Truth and Reconciliation Commission" in South Africa and the "Bloody Sunday" public enquiry in Northern Ireland<sup>14,15</sup>. The deaths of the victims remain in political and criminal dispute and controversy long after the events. There is always a tendency for the victorious side in a war to conduct justice in its own terms, as shown by the Nuremberg Trials of Nazi war criminals in 1945, held jointly by the four allied victors with no German judge<sup>16</sup>.

## **Organ Retention**

Public scandal erupted in 2001 in the UK with a similar process also beginning in the Republic of Ireland. The public enquiry in the UK reported that an individual pathologist at the Royal Liverpool Children's (Alder Hey) Hospital had inappropriately retained many internal organs from post-mortem examinations on children and infants<sup>17</sup>. Parents of the children concerned were outraged at the unlawful retention of organs and some were obliged to carry out multiple funerals. The Chief Medical Officer (for England) carried out a "census" of retained body parts, organs and tissues in NHS hospitals. This revealed many thousands of retained organs in hospitals throughout the UK. A "Retained Organs Commission" was set up to deal with these organs and the related issues, and thousands of enquiries were received on help lines set up in hospitals around the UK. Many enquirers were distressed by the possibility that parts of their deceased relatives were retained in laboratories. Under existing professional guidance, the majority of NHS hospital laboratories had retained tissue blocks and histology slides indefinitely in archives, with a mixed bag around the country of whole organs and larger body parts<sup>18</sup>. Respectable museums and anatomy departments - regulated by the Anatomy Act, 1984<sup>19</sup> - were not immune from holding specimens of doubtful provenance and inadequately documented consent. Some university medical and related departments reported store rooms holding specimens, slides and blocks from long-gone research students and projects. In addition to individual relatives' disputes over the return or disposal of retained human tissues, the affair has illustrated weaknesses in the Human Tissue Act, 1961<sup>20</sup> and current arrangements for post-mortem examinations and consent to the use of human tissues in diagnosis, teaching and research. New legislation has been recommended by the public enquiry and promised by the Government. A public consultation is currently in progress as to the best use of tissue blocks and slides<sup>21</sup>. Stricter regulation of consent for research has already raised concerns that respectable medical and cancer research will be obstructed with no benefit to privacy or data protection<sup>22</sup>.

## **Missing Persons/Bodies Found**

A person reported missing may or may not have died, and relatives may have incomplete or suggestive evidence of what may have happened, or suspicions against particular persons without physical evidence. The classical example is that of the estate agent Suzy Lamplugh who was never seen again after meeting a purported client to view a house. There are strong suspicions that she was murdered but no body has ever been found and insufficient evidence has so far been found to advance the investigation although a suspect, already a convicted prisoner, has been identified.

Unfortunately, many persons go missing each year from their regular lives of domestic contentment and respectable employment. The *National Missing Persons Helpline* has about 2,000 records since 1992. Some are mentally ill and are eventually found; others have deliberately planned to do a "Reginald Perrin" – after the TV character – or a "John Stonehouse" – after the former minister – and have faked their own death or have fled to a new life elsewhere. True cases of "total amnesia" are rare.

The unidentified body found – whatever its cause of death – is a disputed death until identity is established. Clearly, the body was somebody who had a previous

life history and lovers, friends and relations who may be missing him or her. The body may have met a natural or unnatural death. He or she will remain problematic until new evidence is found.

#### **Death Overseas**

Death overseas brings a higher rate of subsequent disputes. Pathological examination may not reach UK standards – although in many countries, especially in North America, Europe and Australasia, standards are excellent – and UK authorities must struggle with inadequate evidence and sometimes an incomplete body returned to the UK. The Helen Smith case was a notorious example of a possible accident, suicide or homicide which left a British coroner with insufficient evidence to determine the course of events. The body returned was incomplete; relatives were dissatisfied with investigations overseas and in the UK; sensational allegations were made amid intense press coverage.

Even after an apparently natural death, a British body may be returned without the internal organs and with a death certificate giving an unsatisfactory cause of death. I have seen overseas diagnoses of "accidental vomit aspiration" in a man with fatal heart disease and "old age" in a woman just sixty years old.

After a road crash or an accidental death, local authorities abroad may have little interest in a thorough investigation of the related evidence and may be reluctant to hold to account their own nationals. British consular officials are of notoriously variable quality and may be uninterested in gathering evidence for the UK coroner and police. In fairness, criminal investigation is not the primary responsibility of a British Consul, but if they cannot assist a UK family with a death abroad, what are they there for at all? A coroner can compel witnesses to attend an inquest in England and Wales but has no powers overseas, although a body falls under his jurisdiction when it lies in – or is returned to – his district.

Increasingly, Britons are pursuing hazardous and ever-extreme sports at locations abroad. Fatal accidents may occur at remote sites and the circumstantial, medical and pathological evidence available may be incomplete. Diving, aviation and mountaineering incidents have peculiar technical factors which require expert assessment usually outside the scope of pathologists at home.

An early example of this was the death of the mountaineers George Mallory and Andrew Irvine on Mount Everest in 1924. They set off but never came back although Mallory's mummified body was found and identified in 1999 at 8,200 m altitude. Controversy remains as to their possible achievement of the summit almost thirty years before Hillary and Tenzing, and on the manner of their deaths<sup>23</sup>.

UK pathologists generally dread the return of a body from overseas. Usually there will be difficulties: the body will be incomplete or decomposed, information will be lacking, important questions of employer's or insurer's liability will be operative and relatives will be severely distressed by the death and the surrounding official difficulties and delays in repatriating the body.

## **Known Death but No Body**

Such cases are a rare variant of the missing person problem. Evidence exists that the person has died but no body is available. With no body, pathologists are of little help but there may be reliable evidence from witnesses or forensic scientific traces that the person has died. Examples can be bizarre and include persons seen to fall

overboard from a ship and be subsequently lost at sea, airmen lost in air crashes, explosions with total disintegration, persons murdered with attempted but imperfect disposal and workmen falling into industrial devices such as furnaces, concrete foundations or production lines.

Captain L.E.G. Oates famously left the tent during Scott's Antarctic Expedition in 1912 saying to his three companions,

I am just going outside and may be some time

Scott recorded in his diary:

He went out into the blizzard and we have not seen him since . . . We knew that poor Oates was walking to his death, but though we tried to dissuade him, we knew it was the act of a brave man and an English gentleman

Oates had suffered catastrophic frostbite to his feet and preferred keeping them frozen to the agony of thawing them out<sup>24</sup>. He had no prospect of recovery without amputation and no relief party was available or feasible in the dreadful circumstances of unseasonably bad weather and insufficient supplies of food and fuel<sup>25</sup>. His body was never found although the bodies of Captain Scott and two companions were later discovered with Scott's famous diary recording their experiences:

These rough notes and our dead bodies must tell the tale . . .

Was the fate of Oates suicide or accidental death? No inquest was held but his estate was bequeathed on the granting of probate to his brother in 1914.

The said LEG Oates having died in the Antarctic Regions . . .

I know this because the transfer is recorded in the deeds of my own house in Leeds, which stands upon land once owned by Captain Oates.

Murder can be prosecuted with no body and an inquest can be held in the absence of the person's remains or with traces reduced to as little as a dental plate or fragments of burnt bone.

### VIPs and Celebrities

Deaths of historical figures and political leaders continue to be controversial after dramatic historical events or assassination. Recent deaths of VIPs and celebrities are liable to dispute owing to sensational media coverage, deception and tampering with evidence, incomplete investigation and conspiracy theorists working on incomplete evidence. Wealthy persons may have many claims on their estate and disputes over the death can complicate division of the spoils. Multiple claimants may appear claiming that the deceased was their biological parent. Parentage can be determined well by DNA techniques but only if DNA specimens from the deceased were preserved. Many celebrities have met peculiar deaths over the years and their stories enter the history of politics, show business and the media. Many pathologists dread dealing with VIP cases as these rarely progress without difficulty.

The answer to these difficulties is for all authorities and professionals involved to play the case "by the book". Evidence should be gathered and examinations made and conclusions reached as if this was not any special person. Any failing by state officials or professionals involved will be found out sooner or later and broadcast by the media and detailed in documentary books advancing conspiracy theories.

## The Media and Disputed Death

Press and broadcast journalists have voracious appetites for news and sensation. The "human interest story" and especially unexpected, dramatic or tragic death are staple subjects of even the most decorous newspaper. From the journalists' angle, the ideal news story includes sex, royalty and death: "Royal found in bed with dead Pope". Even better, if one of the protagonists is crazed by drugs and staggers from the scene and falls to their death from the open window. The ideal political story includes the sudden death of a politician, preferably in salacious circumstances during a general election. "Political leader found dead in brothel!" would thrill any news editor.

The huge public reaction to the sudden unexpected death of Diana, Princess of Wales, was partly due to the intense media coverage on a bank holiday weekend when there was no other news.

Disputed deaths are likely to be reported in the media. Most inquests will have at least a local news reporter disconsolately chewing his pencil and hoping for some titbit of sensation in the otherwise dry proceedings. Local reporters will pass interesting material and photographs on to national colleagues.

The media need "News": That is actual factual content describing interesting, reportable events. They gather news by observing the activities of the emergency services and are often tipped off on an item by the public relations staff of an organisation or an individual with local knowledge. The media will gather photographs, facts and comment from anyone connected with the news. This may involve door-stepping the hapless relatives of a victim or intrusive surveillance. Media agents have invaded hospitals disguised as doctors and have trained long-distance lenses through available windows. Factual information, however confidential, will often leak to the media through informal and uncontrolled channels. Finally, the media have access to the proceedings of Crown and Coroners' courts and may generally report the whole of the evidence verbatim as they wish.

The media work under strict guidelines on decency, sensitivity and accuracy. But as the sensational content of a news item increases, their adherence to the rules will slip<sup>26</sup>.

Professionals dealing with a disputed death must appreciate that anything in their written reports and everything said in the public hearing of the Coroner's Court can and will be reported in the press. Even the walk up to the door of the court may be filmed. It is difficult to look professional and unremarkable with a TV camera trained on your approach. You must not look unduly cheerful or shifty going into an inquest or the Crown Court.

It is the job of the media to report that which is newsworthy in the public domain and in the public interest. Inevitably, the media make errors of judgement in taste and decency and in factual reporting. Media reporting can make a dispute over a death worse by leading the parties to play to the gallery. The widow may have a hysterical outburst on TV; the hospital director may make a defensive speech in an interview to the local radio station. An extended inquest on the victim of a death in custody can become a cause célèbre and may be adopted by political pressure groups, particularly if the victim was from a national, ethnic or racial minority, was mentally ill or was somehow mistreated by police or prison authorities or hospital staff.

## **Defence Approaches to Homicide**

Criminal trials for homicide – murder, manslaughter and infanticide – are a special example of the disputed death. One or more defendants are on trial, indicted with one or more criminal offences. The Crown argue that he did it; the Defendant may plead guilty or not guilty. In a contested trial for an offence of homicide, the events leading up to the death and the evidence of the death itself are the issues in the case. The issues of fact are decided by a jury of twelve ordinary men and women who hear or see the whole of the evidence. Crown counsel argue the case for the prosecution; defence counsel argue the case for the defence. Both sides may call witnesses and show physical evidence as exhibits. The learned judge regulates proceedings, determines points of procedure and law and finally pronounces sentence if the defendant is found guilty. The entire trial is a formalised dispute over the death and how and by whom, if anyone, it was caused.

In criminal trials in the English legal tradition, the standard of proof is "beyond reasonable doubt", although there are some technical exceptions discussed below<sup>27</sup>. In other words, the jury must be sure that the evidence shows that the defendant is guilty of the offences indicted. Moreover, in murder the *mens rea* or wicked intent must be present: the accused intended to kill his victim or at least to cause him grievous bodily harm<sup>28</sup>.

## **Investigation of Homicide**

Police theory and popular drama in the criminal genre show that to convict a person of homicide the following circumstantial facts have to be proven. The accused must have had the means, the opportunity and the motive for killing the victim and therefore did so. But having the means, the opportunity and the motive may be true of more than one individual. Conviction usually needs witness or physical evidence of direct action of the accused upon the victim.

## **Pathological Cause of Death**

Finding the cause of death is fundamental to proving homicide, and cases with suspicious circumstances but no conclusive cause of death are rarely convicted. There should not be reasonable doubt about the cause of death and the defendant's part in causing it.

Forensic pathologists are therefore principally concerned with identifying the cause of death and its causation. The Crown must prove a convincing line of causation between the unlawful actions of the accused and the injuries and death of the victim.

### **Co-factors**

A single stab wound to the chest, witnessed by a passing justice of the peace who hears the accused say, "OK, you bastard, I intend to kill you with this knife", and recorded on street video cameras may seem a simple matter. Real cases commonly involve additional factors which complicate the interpretation of the case. The accused may be mentally ill, intoxicated or under a mistaken impression, such as

that the victim was somebody else or that he had just attacked the defendant's wife or child. The deceased may have been in the last stages of a natural illness with death expected at any time. The deceased may have had a sequence of different injurious incidents during the course of his last and unfortunately eventful day.

## **Degree of Force**

Many cases turn on the degree of force employed in inflicting the fatal injury, whether a stab wound, kicking to the head or manual strangulation. Unfortunately, scientific evidence is generally lacking and the pathologist makes an approximate assessment, on the scale of mild-moderate-severe force, on the severity of the injury compared with experience of previous cases of the same type.

#### **Evidence of Intent**

The prosecution of most offences against the person requires the proof of wicked intent in the accused. As we cannot look into the mind of the accused at the moment of the offence, the court must rely on indicators of intent. These proxy measures may include evidence of planning the incident, motive, the degree of force employed and the reasonableness of force used in self-defence.

Forensic psychiatrists are skilled in the assessment of the mental state of defendants. They may identify whether the accused is fit to plead or to give evidence, and whether any mental illness or abnormality of mind has contributed to the offence.

## **Multiple Accused**

Many cases involve more than one accused person. It is difficult to establish who did what to whom, particularly in fights or freely moving street brawls in which no single witness saw the whole of the action.

## The Defence Pathologist

Defence pathologists are expert witnesses and as such have a dual role which is commonly misunderstood. The first role is to advise their instructing lawyers on the scope and significance of the evidence within their expertise. The second and more strict role is to give expert evidence impartially to the court on the evidence they have seen<sup>29</sup>.

Expert evidence is unusual in that the expert can give his opinion. He will discuss factual findings and then give his opinion upon that evidence: not only, "there was a single stab wound", but also, "this stab wound required substantial force to inflict". The courts are not interested in the opinions of all other witnesses of fact.

Therefore, the job of the defence pathologist before the trial is to investigate and confirm the physical evidence and to assist the defence team in interpreting the evidence in relation to their case. The defence pathologist does not strive to acquit (or indeed to convict) the accused and has no interest in the outcome. His task in the trial is to assist the court to reach a just conclusion. To the despair of defence lawyers, the defence pathologist may, of course, discover, disclose and discuss matters adverse to the defendant's case. Defence experts exist because the balance of advantage both to the individual defendant and to society lies in having clear understanding of the evidence rather than a partisan view of it.

Usual defences to homicide are denial, self defence, provocation, diminished responsibility and action without intent. Denial includes the excuses: I was not there; I have an alibi which proves I was elsewhere; I was there but it was not me. Forensic science examination of trace evidence may, of course, prove presence at the scene or contact with the deceased. Many homicides take place between family members or partners in a marriage or relationship; so presence in the family home and contact are to be expected. Self-defence (or defence of another) depends upon proving that there was an attack from the deceased as the initial assailant. Defensive actions must be reasonable and proportionate given the circumstances. Diminished responsibility covers intoxication and mental illness and is the province of expert forensic psychiatrists. Admission of the action but denial of intent seeks to reduce the case to manslaughter. "I did hit him with the huge rock but I did not intend to kill him!"

It is for the Crown to prove the guilt of the accused person. Some defendants take the passive role: they deny any memory of the events: "I cannot remember what happened". The courts have difficulty where two accused persons remain silent – for example, two parents with a dead baby may say nothing – there is reasonable doubt as to who did what. The long-standing "right to silence" has now been modified so that the silence of the defendant can be taken into account by the jury to the disadvantage of the defendant's interest. "Why will these defendants not explain what happened to their baby, the day he died?".

## **Inquest Pathology**

Apart from the Crown Court in homicide cases, the other most common and important tribunal for the investigation of disputed death is the Coroner's Court in England, Wales and Northern Ireland. Her Majesty's coroner is obliged to hold an inquest in many cases of unnatural death<sup>30</sup>. With some categories of case – such as deaths in custody, hospital mishaps, public transport disasters, generally those deaths with a strong element of protecting the public interest – the coroner sits with a jury. The scope of an inquest is limited by statute and the coroners rules to find the facts touching upon a death: who it was, where it was, when it was and by what means<sup>30</sup>. The coroner must also gather the particulars of the deceased for registration purposes. The coroner is forbidden to enquire into civil liability or to apportion blame to any person or organisation. If criminal liability is present from the outset or becomes apparent later, the coroner adjourns the inquest and refers the matter to the police, Crown Prosecution Service or in some circumstances to the high court. The coroner has limited powers and cannot arrest interested persons, although he can still find contempt of court.

The coroner's proceedings are inquisitorial and legal representatives must refrain from making speeches or reaching conclusions upon the evidence. The value in an inquest lies in answering questions for the relatives in a public court of record. Inquests are frequently reported in the press. Hospital doctors and their directors, police, public agencies and employers often find themselves being held to account in the coroner's court. Embarrassment for failures in safety, conduct and standards of service can be severe.

Many inquests are routine and dull, corresponding to the nature of the majority of deaths. Disputed deaths provide the most interesting inquests and, in the most disputed cases, an inquest can be the most dramatic and emotionally charged experience for its participants. Disputed deaths in the categories of industrial disease,

hospital mishap, road crashes, accidents, suicide, criminal abortion, major disasters and especially deaths in custody are the most important.

After hearing the evidence from persons with relevant evidence to give, the product of an inquest is a written document termed the inquisition. This is prepared by the coroner and represents a summary of the evidence and a verdict on the death. Possible verdicts are not prescribed by law and the coroner can use any form of words he likes. Recorded verdicts number about two dozen<sup>31</sup>. The usual verdicts are natural disease, accident, misadventure, suicide, unlawful killing and open verdict. Misadventure is the unintended consequence of an intended act, such as the death of a boxer who sustained a fatal head injury during a properly organised boxing match. Open verdict means that the evidence was insufficient for the court to reach a more specific verdict: the verdict remains "open", the "don't know" outcome. Verdicts can be qualified by statements such as, "aggravated by lack of care", which introduce concepts of failure by responsible parties.

The scope of an inquest is thus very circumscribed. After the inquest, the coroner files his papers and forgets the case. He may write to responsible authorities to draw their attention to some defect in safety or practice but he has no further powers. Of course, the evidence heard in an inquest can be employed later in civil litigation, public enquiries and criminal courts. A verdict of *unlawful killing* would normally lead to the file of evidence being passed to police and Crown Prosecution Service. Witnesses who say one thing at an inquest and later change their story can have difficulty in being credible in a later court.

The weakness of the coronial service is the marked variation in quality and experience of coroners and their authority to conduct cases as they wish. The consequence is unreasonable variation in practice between districts across the country. The only recourse for aggrieved parties after an unsatisfactory inquest is an application to the Queen's Bench Division of the High Court; not a step to be taken easily or lightly. Coroners are not subject to any supervision or quality audit, except audit of their financial transactions by the Local Authority. Delays can be severe with a lazy coroner and there have been examples of conflict of interest. In small towns, coroners are usually local solicitors, and inevitably they may have previous contact with parties for legal services. In any profession with hundreds of practitioners – be they HM coroners or pathologists – some may be inefficient, some may be ill and some may be drunk.

With the exception of police and pathologists, witnesses appearing at inquests are inexperienced and are usually terrified by the ordeal. This is especially true of junior medical and nursing staff appearing on behalf of a hospital or social or emergency service. The quality of evidence given and the elucidation of a complex medical causation may remain deficient by the end of the inquest.

The interaction between the coroner and his pathologist is crucially important. The coroner has the choice of which pathologist to instruct in each case, although he must consult with the chief officer of police in a case with possible criminal content. Usually, coroners employ a panel of pathologists who organise a service to provide post-mortem examinations. Professor Knight has written the standard work on coroner's pathology<sup>32</sup>. The coroner has control of the ancillary investigations from histology to toxicology and may limit these on cost grounds or to avoid difficulties over organ retention. Increasingly, coroners are taking the narrowest interpretation of their remit and want to know merely whether the death was natural or unnatural and whether an inquest is required. Valuable medical audit data may flow from coroners' post-mortem examinations, with feedback to hospital doctors,

general practitioners, ambulance and fire services, but this is not of interest to the coroner. These medically useful data may be lost to the public interest.

# **Proof and Uncertainty**

#### Rules of Evidence

Pathologists generally have little training in the rules of evidence. This branch of legal studies is arcane and sometimes counterintuitive. Textbooks and short guides are available and should be consulted when in doubt<sup>27</sup>. Many experienced pathologists have experience of sitting in court with the jury sent out while learned counsel debate with the learned judge what is and what is not admissible as evidence. The dispute can seem bizarre to the plain medical man or woman. I have known cases in which photographs were not admissible, forensic science evidence was thrown out and the policeman's notebook entry preferred over expert evidence.

#### **Expert Evidence**

Many expert witnesses have been interrupted by the judge with the bark at counsel, "This is not expert evidence!" or the imperious question, "Is this question within the expertise of this expert?" Expert evidence obviously overlaps with "common sense" in some areas and the best medical experts will try to explain medical matters in terms the jury can understand. The point of difficulty is that the jury themselves can reasonably be expected to assess "common sense" matters from their own knowledge and experience whereas an expert is necessary to explain technical or professional evidence and interpret its causation<sup>27,28</sup>. The usual jury man or woman knows that a punch in the eye causes a black eye and that a bang on the head causes a lump to appear. The jury does not know that there are other explanations for a black eye or how long it takes for the different types of meningeal haemorrhage to develop.

Experts are only credited with expert powers in the narrow field in which they actually have recognised expertise. Expert witnesses can be ridiculed as "liars for hire" – " . . . and what are you an expert in today, doctor?" – owing to the temptation to stray outside one's field and be paid. Pathologists are also doctors and may be asked about medical matters of a general nature. Pathologists should obviously avoid giving their own judgement in specialist matters of anaesthetics, surgery and therapeutics and the like. This said, forensic and hospital pathologists, of all doctors, probably have the widest contacts with doctors in other specialties and in post-mortem examinations are exposed to disease and the effects of therapy in all specialist fields. Pathologists should not hesitate to assist the court when they can: their talent is in explaining to the lay jury or non-medical coroner what the mysterious medical evidence may mean. When the need for specialist expertise arises it should be identified, sought and respected when it arrives.

Comical difficulties can complicate the explanation of physical evidence in sexual matters. Consider cases with allegation of sexual intercourse or fellatio or buggery. Any recourse to the jury's own knowledge or experience will bring sheepish grins and indignant glares. Yet the passage of Object A into Orifices B and or C and or D – I can think of three orifices only – is a common human experience. Sexual injuries exquisitely straddle the uncomfortable boundary between medical expertise and common sense and the science of penetration is woefully inadequate to the need.

Curiously, because paediatricians and police surgeons are usually called first by the police and prosecution, there are few equivalently experienced defence experts in the fields of paediatrics and sexual offences. Clinical doctors in general may have a temperamental disposition to assisting the prosecution of these offenders rather than getting them off. So defence pathologists may be offered cases of offences against the person and courts may struggle to understand the medical evidence in rape and child abuse cases. Some police surgeons are too definite and can be dogmatic about the causation of trivial sexual injuries and some are too vague to be any use; many are excellent.

Expert fields overlap. The classical example which often arises is the interface between forensic science and forensic pathology. A man is murdered with blows to the head from a hammer. The hammer and scene are covered in blood. The head has injuries. The forensic scientist is the expert in weapons and blood staining. The forensic pathologist is the expert in injuries and their causes. A droplet of blood flies out of the field of the pathologist and into the field of the scientist. Each expert should respect the other's end of the business and will usually see each the other's report. But lawyers may try to get each to comment on the evidence of the other and create difficulties.

Experts, however distinguished, experienced and, well, expert, can still be wrong, systematically or occasionally. Professors Gee and Mason show the many ways an expert witness can fail<sup>33</sup>. No-one is immune from error.

#### Standards of Proof

In the criminal courts, the standard of proof is "beyond reasonable doubt"; that is, the jury must be sure that the evidence means the defendant is guilty as indicted<sup>34</sup>. A "reasonable" doubt or a significant alternative possibility should defeat the conviction. In civil courts the standard is "on balance of probabilities"; that is, over fifty per cent likely considering all possibilities. Civil courts are for betting men whereas the criminal courts are for angels and devils. There are technical exceptions. A defendant bears the onus of proof when he brings a special defence such as insanity, diminished responsibility, possession of offensive weapons, or possession of controlled drugs. In other circumstances, the defendant has to raise enough evidence to justify the defence being considered by the jury while the prosecution have the legal burden of disproving the defence offered. Examples of this include drunkenness, provocation, self-defence, duress, automatism, alibi, and belief of consent in sex cases.

The standards of proof are more complex in child-protection proceedings and inquests. In child-protection work, the over-riding duty of the court is the interests of the child. Therefore, the lowest-risk solution is preferred and the mere possibility of abuse can lead to the child being placed into care. In such a case, the sort-of-accused parents are in a sense having to prove their innocence, or at least seeking to show that the risk of them abusing their child is absent.

In an inquest, the general standard of proof is on balance of probabilities, but the verdicts of unlawful killing and suicide require the evidence to prove the verdict beyond reasonable doubt. Given the inquisitorial basis of proceedings, suggestions from witnesses, however unlikely, may be taken into account by the coroner. Witnesses' suggestions of weak value or unlikely probability may yet get into the inquisition and contribute to the verdict. The same case and its evidence can be judged by different standards of proof in different tribunals. Consider a case in which a young child dies after falling down-stairs. He has a fatal injury and other evidence of possible non-accidental injury of mild degree. The child was on the social services "at risk" register for various reasons and the father is accused of causing the death. At the criminal trial, the evidence is not beyond reasonable doubt and the man is acquitted. His wife divorces him and the court accepts on balance of probabilities that he caused the death of the child. His other children are taken into care and he is denied access owing to suspicion of abuse and the over-riding interests of the children. The inquest is resumed on the death of the child and the coroner returns an open verdict. The local paper interprets this as showing that the father caused the death of the child. Different courts have produced different outcomes on the same evidence. Either he did do it or he did not do it; but the state's legal processes have left an unclear determination. If he did not do it, his family life has been wrecked. If he did do it, he has escaped justice and the child's death goes unpunished.

#### **Use of Evidence**

Pathologists must be careful to identify, respect and keep separate facts, hearsay, opinion and conclusions. Poor expert witnesses prepare reports which mangle together hearsay, assumption, speculation, factual findings and opinion to draw conclusions without logical interpretation or consistency. Good pathologists must strive to be analytical: to receive the alleged history, to investigate factual findings, to form defensible opinions upon the facts, and to produce clear conclusions on the questions at issue.

Hearsay technically is a prior statement – prior to the court proceedings – tendered in evidence to prove the truth of its contents. English law has for centuries been preoccupied with the rule against hearsay and exceptions to the rule<sup>35</sup>. The courts prefer "real" evidence direct from witnesses of what someone saw, not what they were told by someone else. In practice, hearsay is when someone else tells you: this usually includes the instructions from the police or one's instructing solicitors. Hearsay is signalled by the words alleged or allegedly. "It seems the vicar was in the brothel when he keeled over . . ." is less clear than "Madame Smith alleges that the vicar was in her brothel when he collapsed . . ." The output from computers, instruments and the like is regarded as real evidence and not hearsay, so long as the device was working properly.

A *fact* is a physical finding made by the pathologist using his own eyes or the documented evidence of some other professional's report or findings which can be checked with its source. A toxicology report is factual; its contents can be checked and discussed with the toxicologist.

Opinion is the interpretation of facts made in the setting of the alleged case by the pathologist or expert witness<sup>28</sup>. Whether the defendant is guilty as proved is the "ultimate issue" for the court and in a criminal case this is decided by the jury who hear the whole of the evidence. An expert will usually have seen only part of the evidence to give his opinion upon, so he should avoid giving his view on the ultimate issue or making assumptions. If opinion is contingent upon other evidential possibilities, each possibility must be explained fully.

A *conclusion* is a statement which summarises the result of a piece of analysis and usually represents the considered answer to one of the questions at issue in the case.

A pathologist's report should present hearsay, facts, opinion and conclusions in that order in separately identified sections. It is always difficult to be completely clear on complex matters and many reports can be improved by further work. Even the best pathologists find that something in their report has been misunderstood or could have been made even clearer.

Proof is rarely absolute; pathologists more usually are asked to decide whether physical evidence is or is not consistent with some suggested causation. For A to be consistent with B does not mean that B definitely caused A. Other causes – C, D and so on – may also be consistent with A. In many ways, the negative is the more useful finding: "A was not consistent with B" is a useful contribution.

#### **Expected Ranges**

Pathologists are at their worst when trying to fit dynamic problems into definite ranges. The notorious example is time of death. A case may have good evidence showing the deceased last seen alive and later found dead at definite times. Accused persons may have reliable alibis for parts of the period at issue. Pathologists may be asked to state with precision when the person died within the possible time span of the incident. Unfortunately, scientific methods for time of death are imprecise and only approximate ranges can be given. Scientific methods may give statistically likely ranges but unfortunately the criminal standard of proof is "beyond reasonable doubt" and doubt is part of estimates of probability. This problem is avoided in civil litigation where probability is the valid test. Avoidance of the fallacy of precision is a test of pathological character.

# **Investigation of a Disputed Death**

#### **Instructions**

Inevitably, pathologists have to be involved in investigating a disputed death. The instructions may come from the state (HM Coroner, police, Crown Prosecution Service, public enquiry) or from the legal representatives of an aggrieved party. It is always unwise to accept instructions directly from an interested party privately. Individuals may not understand the rules of the game and may be cross when the report delivered does not support their case. They may refuse to pay for services.

The investigational process belongs either to the state or to the instructing party. The report is rendered unto who is paying. As explained above, experts have duties to the court and to justice which over-ride those of the paying party in the case. It is fundamentally important to have clear and reasonable instructions before acting in a case. The expectations of the instructing party should be realistic. Sometimes, instructions include misleading accounts of events or set questions in biased ways. Many pathologists will act pro bono publico on occasion, and will usually spend some time explaining informally the scope of expert evidence to litigants in person or distressed relatives without charge or obligation.

#### **Approach**

The open mind is most successful when focussed upon the evidence. Assumptions at the outset may not be true and the whole of the available evidence should be

reviewed. The issues which determine the outcome should be analysed and concentrated upon. Common ground can be accepted and then receive less attention.

#### Investigation

The investigating pathologist should gather evidence of the history of the deceased, the preceding events and carry out a post-mortem examination. Second postmortem examinations have limitations, especially if organs are missing or the body is deteriorated. Close consultation with the first pathologist is essential but may not be possible. The pathologist must collect the factual background to the incident or illness and examine each record or statement against a chart of the "time line" to discover inconsistencies. Hospital notes commonly show deficiencies with undated and unidentified entries. The experienced medical negligence lawyer turns first to the nursing record, which is usually legible, comprehensive and includes physiological measurements such as temperature, pulse and blood pressure. Notes by doctors are often less satisfactory. Classical examples include the note of the "successful" operation followed by blank pages and the determination of death in the medical notes, compared to a complete record in the nursing notes over four post-operative days of rising pulse, falling blood pressure and repeated notes of "Doctor called, did not come" and interventions by angry relatives.

In post-mortem work, essential ancillary scientific investigations – biochemical, toxicological, microbiological, haematological and more rarely genetic – must be thought of, carried out on good specimens and interpreted with the assistance of expert colleagues.

In complex clinical matters, the case should be discussed informally and confidentially with a colleague in the field but unconnected with the case. Other expertise may be consulted when there is overlap between disciplines. In the worst post-operative disasters, for example, the case may span a branch of surgery, anaesthetics, intensive care medicine, microbiology, metabolic medicine and pharmacology.

It is invaluable for pathologists to visit the scene of a disputed incident, to see any vehicles or alleged weapons and to examine all relevant photographs.

Pathologists' evidence may become admissible in a wide variety of tribunals in addition to the usual Coroner's Court or Crown Court in England and Wales. In Scotland, the Procurator Fiscal may cause a Fatal Accident Enquiry to be held in the Sherriff's Court. Disputed cases may go to the High Court or be subject to a public enquiry. In the UK, "Confidential Enquiries" have been set up routinely to enquire into cases of maternal death, suicide, deaths in infancy and stillbirths. Occasional research projects are conducted nationally, on such subjects as road crashes and drowning cases.

The product of the pathologist's or expert's death investigation is the report. This should follow established forms and, above all, must keep separate sections repeating the instructions, repeating the allegations, history or hearsay evidence, the physical and factual findings and opinion upon those findings. Conclusions should be written to answer, so far as is possible, the questions or issues in the case. The degree of certainty or uncertainty must be clearly stated and the possibility of additional investigations producing useful evidence must be assessed. There are various prescribed "expert declarations" attesting to the truth of the report which one places above one's signature.

After producing a report, the pathologist must remain available to discuss it and to explain and clarify the issues and findings to the recipients of the report. Pathologists must attend and give evidence in person to the various tribunals when so instructed. Their oral evidence must closely follow their written report, but technical matters must be explained in lay terms so that the findings and issues are understood.

#### **Outcomes**

The ideal outcome to a death investigation is a satisfactory explanation of the death and the issues raised. The identity of the body is confirmed, the cause of death is agreed, the contribution of the defendant, the hospital, the police or the airline is proved by the evidence. Inconclusive cases are the worst but, sometimes, the evidence available is not sufficient to answer the questions remaining.

Official tribunals will produce their determination: an inquest verdict, a criminal conviction or acquittal, an enquiry report. The press will report the findings. Appeal processes or political agitation may persist and questions of civil liability make take years to pursue through the courts.

Inquests and criminal courts do have weaknesses of process and sometimes fail to satisfy the relatives or the public interest. Inevitably some cases will remain unsatisfactory. There is a strong history of miscarriages of justice in the UK, some historical, some current, but many very serious<sup>36</sup>. As David Jessel, former presenter of *Rough Justice* and now a commissioner for the Criminal Cases Review Commission, has indicated, if only one per cent of prisoners have not been rightfully convicted, then a large number of innocent people are in prison.

Although no amount of effort in a disputed death can bring the deceased person back to life, efforts to resolve outstanding disputes are beneficial in serving the continuing interests of the surviving relatives and society as a whole. Most relatives will sooner or later come to resolution of their grief and acceptance of the evidence making a good death a valuable part of life. Settling disputes over the dead is an essential function of civilised society.

Pathologists can help the dead and their relatives to rest easy. If they undertake reasonable instructions, diligently examine the available evidence and reach soundly based conclusions presented in lucid reports, they will have succeeded in their task as public servants, judicial agents, members of the medical profession and of humanity.

# References

- 1. Picknett N, Prince C, Prior S. Double standards: The Rudolf Hess cover-up. London: Time Warner Paperbacks, 2001.
- 2. Thomas G, Dillon M. The assassination of Robert Maxwell: Israel's superspy. London: Robson Books, 2002.
- 3. Lock M. Twice dead: Organ transplants and the reinvention of death. Berkeley: University of California Press, 2002.
- Gray C. Book: Twice dead: organ transplants and the reinvention of death. (Book review). BMJ 2002;324:1401.
- 5. Beevor A. Berlin: The downfall 1945. London: Viking, 2002.
- 6. Convers Nesbit R. Missing believed killed. Stroud: Sutton Publishing Ltd, 2001.
- 7. Rutty GN (Ed.) Essentials of autopsy practice, vol 1. London: Springer, 2001.

- 8. Dorries CP. Coroner's courts: a guide to law and practice. Chichester: John Wiley and Sons, 1999.
- 9. Berry PJ. Intra-alveolar haemorrhage in sudden infant death syndrome: a cause for concern? (Editorial). J Clin Pathol 1999;52:553-4.
- Green MA. A practical approach to suspicious death in infancy a personal view. J Clin Pathol 1999;51:561–3.
- 11. www.copwatcher.freeserve.co.uk/custodydeaths/ (cited 2002).
- 12. Walters TC, O'Connell MA. A Guide to the Police and Criminal Evidence Act 1984. London: Financial Training Publications, 1985.
- 13. http://www.thelockerbietrial.com/ (cited 2002).
- 14. www.doj.gov.za/trc/ (cited 2002).
- 15. www.bloody-sunday-inquiry.org.uk (cited 2002).
- 16. Conot RE. Justice at Nuremberg. New York: Harper and Row, 1983.
- 17. Report into the Royal Liverpool Children's Hospital. London: Department of Health, 2001.
- 18. Report of a census of organs and tissues retained by pathology services in England. London: Department of Health, 2001.
- 19. Anatomy Act, 1984.
- 20. Human Tissue Act, 1961.
- 21. Retained Organs Commission. Annual Report April 2001-March 2002. London: The Commission, 2002.
- 22. Furness PN. Useful things you cannot do with "surgical waste". Acp News 2001; Spring: 24-26.
- Holzel T, Salkeld A. The mystery of Mallory & Irvine. Fully revised edition. London: Pimlico, 1999.
- 24. Preston D. A first-rate tragedy: Captain Scott's Antarctic expeditions. London: Constable, 1997.
- Solomon S. The Coldest March: Scott's fatal Antarctic expedition. New Haven and London: Yale University Press, 2001.
- 26. Belsey A. Journalism and ethics: can they co-exist? In: Kieran M, editor. Media Ethics. London: Routledge, 1998;1–14.
- 27. Aquino T. Essential evidence. 2nd ed. London: Cavendish Publishing, 2000.
- 28. Gee DJ, Mason JK. The law of evidence. In: The courts and the doctor. Oxford: Oxford University Press, 1990;63.
- 29. Gee DJ, Mason JK. The courts and the doctor. Oxford: Oxford University Press, 1990.
- 30. Dorries CP. Appendix 5: Coroner's Rules 1984. In: Coroner's courts: a guide to law and practice. Chichester: John Wiley and Sons, 1999;327–60.
- 31. Dorries CP. No definitive list of verdicts. In: Coroner's courts: a guide to law and practice. Chichester: John Wiley and Sons, 1999;200-1.
- 32. Knight B. The Coroner's Autopsy: a guide to non-criminal autopsies for the general pathologist. London: Churchill Livingstone, 1983.
- 33. Gee DJ, Mason JK. Failure of the expert witness. In: The courts and the doctor. Oxford: Oxford University Press, 1990;143–59.
- 34. Aquino T. Burden and standard of proof. In: Essential evidence. 2nd ed. London: Cavendish Publishing, 2000;1–12.
- 35. Aquino T. The rule against hearsay. In: Essential evidence. 2nd ed. London: Cavendish Publishing, 2000;99–109.
- 36. Kennedy L. Thirty-six murders and two immoral earnings. London: Profile Books, 2002.

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#### Introduction

Maternal deaths in the United Kindgom (UK) are uncommon. The overall rate of maternal death has declined with a corresponding decrease in the infant mortality rate. Maternity services in the UK are sophisticated in comparison to less developed countries where the maternal mortality rate is still very high. Given the increasing inequalities in health care between the developed and undeveloped world, the maternal mortality rate is likely to increase. Even in the UK, immigrant groups are still over-represented in maternal death statistics1 with the risk in these groups being three times greater than that of white women<sup>2</sup>. Mortality is particularly high in countries afflicted with a high incidence of life-threatening infections, an important new development being the increasing incidence of HIV infection and its effects on the development of associated infections<sup>3</sup>. The aim of this chapter is to provide an overview of some of the more important problems of pregnancy which may present as maternal death, with some emphasis on issues which are likely to increase in frequency over the years to come. However, this remains an overview of the subject and thus for a more detailed consideration into this area specialist texts should be perused, since one short chapter cannot possibly address the myriad problems of complications of pregnancy<sup>4</sup>.

#### **Definitions**

Maternal death is defined by the International Classification of Diseases, Injuries and Causes of Death (ICD9) as "the death of a woman while pregnant or within 42 days of termination of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes". Maternal deaths in the UK are investigated as part of the Confidential Enquiry into Maternal Deaths. This enquiry, established in 1952, is rather broader in its terms of reference that the ICD definition of maternal death. The Confidential Enquiry also includes late maternal deaths and fortuitous deaths. The Enquiry is a triennial survey, the latest report for 1997–1999 being published in 2001<sup>2</sup>. The definition of maternal death for this enquiry is shown in Table 4.1.

Direct death	The death can be directly ascribed to complications of the pregnancy (pregnancy,
	labour and puerperium) and from interventions and treatment thereof.
Indirect death	The death occurs as a consequence of the pregnancy exacerbating a pre-existing
	medical condition or a medical condition developing in pregnancy but not directly
	attributable to that pregnancy.
Late maternal death	Death occurs between 42 days and one year post-delivery from conditions that
	are due to direct or indirect causes.
Fortuitous death	Death is due to causes unrelated to the pregnancy or the puerperium.

Table 4.1 Definition of maternal death from the Confidential Enquiry into Maternal Deaths, 2001<sup>2</sup>.

Maternal deaths must always be reported to the Director of Public Health and usually to H.M. Coroner. Some cases still occur where the pathologist is the first doctor to discover that the deceased was pregnant. Such cases are now uncommon since the passage of the Abortion Act, 1967<sup>5</sup>. Concealed pregnancy still occurs, usually in very young women or in women with psychiatric disorders, but maternal deaths in these cases are still uncommon.

#### **Causes of Maternal Death**

Three hundred and seventy eight deaths were considered within the remit of the last confidential enquiry<sup>2</sup>. Although this was similar to the previous report (376 deaths, 1994–1996) there was a slight downwards trend in the number of reported deaths, with the present rate for the triennium of 11.4 deaths per 100,000 maternities. This is compared to the rise observed during the last enquiry but shows a dramatic difference from the first enquiry (67.1 deaths per 100,000 maternities, 1952). This previous rise had been attributed to improved case ascertainment.

When one considers both direct and indirect death, the mortality rate for direct deaths was reduced at 5.0 deaths per 100,000 maternities whereas indirect deaths were up at 6.4 deaths per 100,000 maternities. It is most difficult to ascertain statistics in fortuitous deaths: in such cases, these may not be recorded as being a death in pregnancy or the puerperium since that pregnancy is not related to the cause of death. In subsequent enquiries, the age range may well need to be increased with a rise in the pregnancy rate amongst very young girls.

The commonest causes of direct maternal death identified in the enquiry are shown in Table 4.2. The first five of these categories accounted for the majority of deaths. The commonest cause of direct maternal death is pulmonary thromboembolism from deep vein thrombosis (DVT), accounting for 33% of direct maternal deaths. Pregnancy induces a hypercoagulable state and as the uterus enlarges, pressure on the pelvic and deep leg veins increases, in addition to decreasing mobility as the pregnancy progresses. The risk of DVT increases with advancing maternal age and parity. DVT can occur at any stage during pregnancy but the risk increases as the pregnancy progresses, so that over two thirds of cases actually occur post-partum<sup>6</sup>.

Congenital heart disease in pregnancy poses a difficult management problem and may result in premature delivery, either induced or spontaneous. Operative intervention may be necessary, which may in turn endanger the pregnancy itself. Epilepsy, too, is difficult to manage; the frequency of fits may increase in pregnancy, but the benefits of adding or increasing anti-epileptic drugs must be weighed against

**Table 4.2** The commonest causes of direct and indirect maternal death identified in the Confidential Enquiry into Maternal Deaths, 2001<sup>2</sup>.

Direct	
Pulmonary thromboembolism (PTE)	35
Ectopic pregnancy	17
Pregnancy-induced hypertension	15
Sepsis	14
Amniotic fluid embolism	8
Haemorrhage	7
Uterine rupture	3
Fatty liver of pregnancy	2
Anaesthesia	3
Fatty liver	1
Indirect	
Cardiac causes	35
Other indirect	75
Psychiatric causes	15
Indirect malignancies	11

the risks to the foetus from the drugs or from the risks of the effects of a grand mal fit. Epileptic fits may be difficult to distinguish clinically from an eclamptic fit, the latter being uncommon, but still not unknown in modern obstetric practice. In most instances, a history of epilepsy will be available but this may not necessarily aid the specific diagnosis in the first instance. Sudden death is well recognised in epilepsy and pregnant women have a higher risk of pregnancy complications<sup>7,8</sup>.

Late maternal deaths may not be recognised as being such. These deaths may be due either to direct or indirect causes or may be fortuitous. Late maternal death from direct causes is the least common and is usually due to thromboembolism (which still poses a significant risk in the puerperium), or amniotic fluid embolism. Some late deaths occur because the woman had received intensive care which has delayed the death, even though the condition leading to the collapse was severely life-threatening in the first instance. Indirect causes constitute approximately half the late maternal deaths recorded in the last enquiry. The majority of these deaths were related to cardiovascular causes including puerperal (peripartum) cardiomy-opathy, and two cases of thromboembolism in post-partum women prescribed the oral contraceptive pill.

Other uncommon causes of late death include neoplastic disease, where the pregnancy may have masked the disease or altered its course, cystic fibrosis and asthma.

A relatively large group of maternal deaths includes deaths due to suicide and substance overdose. There were 28 late deaths recorded in this group between 1997 and 1999, showing an increase from the previous report. This again was due to improved data-collection methodology. Suicide in pregnancy and the puerperium is uncommon despite the incidence of psychiatric illness in childbearing women being higher than in the rest of the population. It may be that the presence of an infant may have a protective effect against suicide. The childbearing woman often shows warning signs of depression or other psychiatric features, but this is not always the case. The last enquiry included six deaths from substance misuse and four alcohol-related deaths. Given the prevalence of substance and alcohol misuse, these constitute a surprisingly small number of cases.

There were 90 fortuitous deaths recorded by the last enquiry, including 61 late fortuitous deaths. These deaths are not included in the ICD definition of maternal death, since, by that definition, the death is unrelated to the pregnancy. The commonest causes of fortuitous death were road traffic accident (8 deaths), murder (8 deaths) and neoplastic disease (6 deaths) where the disease progression was thought to be unrelated to the pregnancy. Domestic violence is a problem in most societies, but homicide as a mode of death in the pregnant woman remains uncommon in the UK. This is a commoner mode of death in the USA and probably reflects the higher homicide rate in general.

# **Investigating the Maternal Death**

In the UK, each Health Authority has assessors for obstetrics, anaesthetics, pathology and midwifery. In addition to this there is a number of national assessors who collate the overall statistics and compile the triennial enquiry report. A maternal death should be reported to the local Director of Public Health, this responsibility lying with the consultant, midwife or general practitioner who cared for the woman. Local policies should be in place for appointing a coordinator to gather all information regarding the death.

The pathologist has a key role to play in investigating the maternal death. Ideally, all maternal deaths should be referred to an experienced pathologist, who may not necessarily be the local pathology assessor. This may not always be practicable, so that it is important that the general histopathologist is aware of at least the basic procedure required properly to investigate a maternal death. During their working life most pathologists will never encounter a maternal death. For those who do, carrying out the autopsy and ancillary investigations is not difficult provided that there has been a reasonable amount of thought beforehand regarding the type of problems which may be encountered in the pregnant woman. This requires a certain amount of preparation. In cases of difficulty, guidance may always be sought from more experienced personnel.

Prior to starting a maternal death autopsy, the most important considerations for the pathologist are set out below. Specific recommendations can be found in Appendix A to Chapter 18 of the confidential enquiry<sup>2</sup>.

- Appropriate discussion with clinical colleagues before the autopsy, if necessary, to assist in interpretation with the clinical notes. This is particularly important in anaesthetic deaths, which are frequently complicated and may result in few pathological features. Equally, few pathologists are widely experienced in obstetric practice and interpretation of the clinical management of pregnancy requires expert assistance.
- Reading the clinical notes thoroughly before the autopsy (including fluid balance) or any local guidelines into the management of hypertensive disorders of pregnancy. This should alert the pathologist to the possible cause(s) of death, thereby allowing appropriate planning of investigations. This is important since many mortuaries do not keep materials such as viral transport medium, blood culture bottles or a variety of blood sample containers. Specimens may have already been sent to the laboratory, but the results may not be available in the notes. Since ante-mortem samples are preferable to post-mortem samples,

attempts should be made to discover these results before the autopsy. If the pathologist considers that a particular technique may be required with which he may not be familiar, a cautious approach allows the pathologist time to revise that technique using an appropriate text<sup>9</sup>.

• Reviewing all organs or tissues that have been resected prior to death.

• Taking appropriate samples for ancillary investigations: these include histology, microbiology, toxicology and, occasionally, neuropathology.

# **Retention of Material at Autopsy**

Maternal deaths are often the subject of a coroner's inquiry. In these cases, section 20 (4) of the Coroners Act, 1988<sup>10</sup> and sections 11 (2 and 4) of the Coroners Rules, 1984<sup>11</sup> govern the retention of material at the autopsy. Some cases will fall outside the jurisdiction of the coroner. Retention of material at these non-coronial "consent" ("hospital") autopsies is governed by the Human Tissue Act, 1961<sup>12</sup>. If in doubt, clarification should be sought either from H.M. Coroner (or the local equivalent) or by referral to a specialist text<sup>13</sup>.

Following the Alder Hey Enquiry<sup>14</sup>, there has been considerable disquiet concerning the retention of material at autopsy. The Royal College of Pathologists has issued guidelines concerning retention of material at autopsy<sup>15</sup> and there are regular updates from the Department of Health. Each hospital should have a local policy in place and many will have named individuals dealing with documentation related to tissue retention, storage, disposal or returning material to relatives for the purpose of returning to the body. As with any matter related to autopsy, the pathologist should record the exact nature of material retained in a case, including microbiological and toxicological material. This is usually done by means of a pro forma which is acceptable to all parties. An example of such a form is shown in Fig 4.1. The coroner or relatives must be kept informed at all times of any material retained at the autopsy. The coroner has no power to authorise retention of material for any purpose other than ascertaining the cause of death. Therefore, relatives' permission must be sought if the pathologist wishes to retain any material for research or other purposes.

#### **Direct Maternal Deaths**

#### **Thrombotic Problems**

The commonest cause of death at any stage in pregnancy is pulmonary throm-boembolism (PTE), with 31 deaths in the last enquiry. This equates to a rate of 1.45 deaths per 100,000 maternities. This showed a fall in the number of deaths from PTE compared to the previous report. The principal risk factors for PTE are the age of the patient, long-haul air travel, a family or previous history of PTE, immobility, maternal weight and use of oral contraception. In addition to this, over the last decade, a group of patients have been recognised who have hereditary disorders predisposing to recurrent thromboembolism, eclampsia and foetal loss. Antiphospholipid antibodies can be detected in a number of conditions including

# DIVISION OF FORENSIC PATHOLOGY UNIVERSITY OF LEICESTER

#### Retention of Tissue and Body Fluids Form

PATHOLOGIST: NAME OF DECEASED:
PLACE OF EXAMINATION: DATE OF POST MORTEM:

The following material has been retained (approximately 2g of each) for the purpose of microscopic examination for the cause of death.		The following whole organs have been retained for the purpose of examination for the cause of death.					
SITE	YES	NO	NUMBER	ORGAN	YES	NO	EXAM PLACE
Brain				Brain :			
Meninges				Spinal cord			
Spinal cord				Eyes			
Heart				Heart			
Right Lung				Right Lung			
Left Lung				Left Lung			
Liver				Liver			
Spleen				Right kidney			
Right kidney				Left kidney			
Left kidney				Bone			
Prostate					0	Manager Car Ma	
Ovary				The following body fluids have been retained for the purpose of cause of death.			been retained for the
Uterus							
Testes							
Adrenal				SITE	YES	NO	SENT TO?
Thyroid				Blood			
Salivary gland				Urine			
Pancreas				Stomach content			
Bone				Cerebral spinal fluid			
Skin				Vitreous			
Thymus				Bile			
Other							

#### OTHER SAMPLES/TESTS

EXAM TYPE	YES	NO	SAMPLE	SENT TO?
Haematology				
Clotting studies				
Metabolic studies				
Genetic studies				
Microbiology				
Virology				

**Fig. 4.1** An example of a pro forma used to inform H.M. Coroner and the relatives of materials retained at an autopsy.

rheumatoid, lupus and other collagen disorders. There is a group of women who do not have lupus but who have antiphospholipid antibodies and who suffer recurrent miscarriages. These women have Primary Antiphospholipid Syndrome, also known as Hughes' Syndrome<sup>16,17</sup>, which can be detected using clotting and immunological assays. Reducing the risk of coagulation is possible, although anticoagulation in the pregnant woman poses a problem. Warfarin and other coumarin anti-coagulants are teratogenic and their use is contraindicated in pregnancy. Aspirin or fractionated or unfractionated heparins will be used where required<sup>18,19</sup> and a careful check should be made in the obstetric notes to ascertain whether anti-coagulation therapy was prescribed.

Heritable coagulopathies are a major cause of maternal thromboembolism, the commonest including antithrombin III deficiency and protein C deficiency. As well as increasing the risk of thromboembolism up to eight times, there is also an increased risk of pre-eclampsia and intrauterine growth retardation. The risk of stillbirth is also increased. Any patient with a known coagulopathy should be offered prophylactic anti-coagulant treatment and this therapy must continue into the puerperium, given the risk of PTE in this period<sup>19</sup>.

Sudden death due to PTE may rarely present as a suspicious death: the principal author has investigated two such cases. In dealing with such a death, the pathologist should be aware that the woman or her associates might not have been aware that she was pregnant. As such, the pathologist may be the first doctor to become aware of the pregnancy.

Cerebral thrombosis is a rare complication of pregnancy (four cases 1997–1999) and may be associated with a hypertensive disorder<sup>20</sup>. The risk of cerebral infarction increases in the puerperium, and amniotic fluid embolism is a rare cause of stroke (see below)<sup>21</sup>.

#### **Hypertensive Disorders of Pregnancy**

Fifteen patients were reported to have died from hypertensive disorders (7.5 per million maternities). Routine antenatal screening should alert the clinician to potential hypertensive disorders, although these can develop very rapidly. However, some women default on appropriate ante-natal care, including women with concealed pregnancy.

Primary hypertensive disorders of pregnancy encompass both pre-eclampsia and eclampsia, but hypertension is not necessary for the diagnosis of pre-eclampsia since the condition represents a spectrum of disorders, and blood pressure can fall during early pregnancy. The incidence of eclampsia has declined over succeeding decades<sup>22</sup>, but this is not a reason for complacency. The leading causes of death in this group of conditions in the last enquiry were intracranial haemorrhage and pulmonary problems, including pulmonary haemorrhage and oedema. The causes of death were thus grouped into cerebral (intracranial haemorrhage, subarachnoid, infarct or oedema), pulmonary (adult respiratory distress disorder and oedema), hepatic (rupture and failure/necrosis) and others. The HELLP syndrome (haemolysis, elevated liver enzymes, low platelets) and hepatic and renal failure can occur. A sub-capsular haematoma may form in the liver, this being prone to rupture. Appropriate histological examination is essential in the post mortem diagnosis and investigation of hypertensive disorders. The placental bed should be examined for atherosis of spiral arteries. The kidney should be examined for glomerular fibrin

deposition in cases of disseminated intravascular coagulation (DIC) (see Chapter 5) and the liver should be examined for periportal infarction.

Many of the symptoms and signs of HELLP Syndrome may overlap with acute fatty liver of pregnancy (AFLP). Whilst not a primary hypertensive disorder per se, it typically occurs in the third trimester, presenting with a picture of nausea, epigastric pain, jaundice, a bleeding diathesis and hypertension. This condition may be readily diagnosed at autopsy by use of a frozen section/Oil Red O stain: macroscopically, the liver may have a greasy texture and yellow sheen.

#### **Haemorrhagic Disorders of Pregnancy**

The main problems are placenta praevia, placental abruption (which is mainly a third-trimester problem) and post-partum haemorrhage. Placenta praevia may be particularly dangerous if associated with a uterine scar. Placental abruption can be difficult to diagnose on post mortem examination alone, even if the placenta is available for examination. Most specialist texts report that this is a clinical, not pathological diagnosis and therefore close communication with the attending clinicians is recommended. The blood clot easily becomes detached from the placenta. A careful note should be made of the site of placental attachment and a search made for a scar from previous caesarean section.

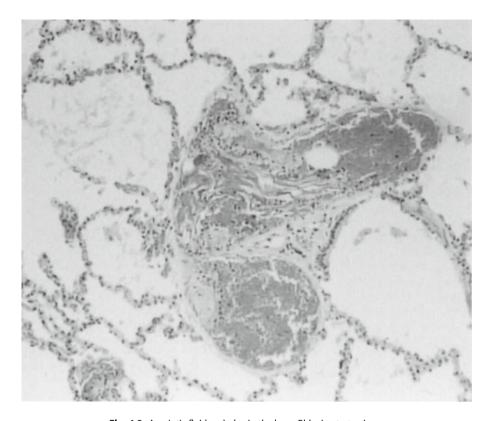
Post-partum haemorrhage accounted for six deaths reported to the most recent enquiry. If an obstetric procedure has been performed, the genital tract should be examined for operative problems such as lacerations, bleeding foci or retained products. It is important to examine the notes for recent blood counts and coagulation studies.

#### **Amniotic Fluid Embolism (AFE)**

This condition accounted for eight direct deaths in the last enquiry. The risk of AFE increases with age with a dramatic rise in cases in those aged 35 and above. Other risk factors identified included the presence of antenatal complications, induction or augmentation of labour and obstetric intervention.

Whilst being rare in relation to the number of successfully completed pregnancies, AFE is a major cause of non-haemorrhagic obstetric shock, together with PTE, acute uterine inversion and genital tract sepsis<sup>24</sup>. When this condition occurs, collapse is usually very rapid and is manifest by acute onset of breathlessness, cardiovascular collapse and acute coagulopathy (DIC). There is recent evidence that some of the haemodynamic alterations in AFE may be due to endothelin-1 expression<sup>25</sup> and that this causes acute lung injury and left ventricular dysfunction<sup>26</sup>. Anaphylaxis has also been hypothesised to play a part in the pathology of AFE: one study has shown an increased number of pulmonary mast cells in women dying of AFE in comparison to control groups<sup>27</sup>.

Unfortunately, there is no specific diagnostic test for AFE although serological methods have been attempted<sup>28</sup>. Amniotic fluid embolus is usually diagnosed by signs and symptoms and their close temporal relationship to labour and delivery. These signs usually appear within an hour of delivery and when AFE occurs, the prognosis is poor. Amniotic fluid embolus can be satisfactorily diagnosed at autopsy using a modified phloxine tartrazine stain on sections of lung<sup>29</sup> (Fig 4.2). The use of immunohistochemical staining for cytokeratin may work to similar



**Fig. 4.2** Amniotic fluid embolus in the lung. Phloxine tartrazine.

effect<sup>30</sup>, and recent attempts have been made to stain for AFE in pulmonary sections using antibodies to glycoproteins found in meconium and amniotic fluid<sup>27</sup>.

#### **Ectopic Pregnancy and Abortion**

Deaths from illegal abortion are now virtually unknown in Britain since the Abortion Act 1967<sup>5</sup>. With the decline in illegal abortion, the rate of genital tract sepsis, air embolism and post-abortion haemorrhage has declined sharply. Really, the only instances of illegal abortion now occur when the procedure takes place in premises not licensed to perform this procedure. Air embolism is now very uncommon, but was common prior to the Abortion Act 1967. The aetiology of air embolism was usually due to the introduction of abortifacients into the uterine cavity, including soapy water, with escape of gas into the uterine veins. Inexpert instrumentation in an attempt to achieve abortion had the same effect.

Ectopic pregnancies occur almost exclusively in the first trimester and therefore fall within the category of early pregnancy deaths. Ectopic pregnancy can be difficult to diagnose. First, one has to suspect that the patient may be pregnant. The diagnosis has been improved since the introduction of quantitative hCG testing and transvaginal ultrasound, which should allow a diagnostic rate for ectopic pregnancy approaching 100%<sup>2</sup>. It can be difficult to elucidate symptoms in the woman

whose first language is not English and who may have relatively little experience of the health care system in the UK. It is now more common for attempts to be made at tubal salvation rather than excision of the whole mass with the fallopian tube. This is not without risk and is not always suitable in advanced cases.

Abortion may be spontaneous or induced for a variety of reasons under the Abortion Act 1967. Therapeutic abortion is relatively safe and with appropriate care, post-procedure sepsis is now a relatively uncommon problem in developed societies.

#### **Genital Tract Sepsis**

There were 14 deaths from sepsis recorded in the most recent enquiry. The majority of these cases was from sepsis after surgical procedures, including caesarean section, and puerperal sepsis. The most serious and life-threatening organism was that of beta-haemolytic *Streptococcus pyogenes* (Lancefield Group A). Lancefield Group C and G organisms may also cause serious clinical syndromes and Group B may cause neonatal septicaemia and meningitis. Prolonged rupture of membranes is a significant risk factor for the development of serious sepsis<sup>23</sup>. Genital tract sepsis was a major problem before appropriate infection control measures were instigated early in the twentieth century. Sepsis is still a significant problem, particularly where there is poor obstetric care in less developed societies. Sepsis may be contributed to by genital mutilation of women where this practice is still frequently practised, albeit illegally: there may be ongoing infection in the woman who has suffered this type of trauma and mechanical obstruction of labour is a common problem, particularly in the primigravida<sup>31,32</sup>.

#### **Anaesthetic Deaths**

Deaths associated with anaesthesia may occur in conjunction with a number of other pregnancy-associated problems. The death is not necessarily due to substandard anaesthetic care. Pre-existing cardiac disease and pulmonary hypertension constitute difficult anaesthetic problems, as does anaesthesia in the pre-eclamptic woman. Cases of haemorrhage and sepsis were also implicated in several anaesthetic-related deaths in the latest enquiry. Since obstetrical emergencies occur very quickly, leading to rapid collapse, appropriate anaesthetic care is vital.

Anaesthetic-related deaths are frequently difficult to investigate from a pathological point of view and underline the necessity of seeking assistance from a suitably qualified anaesthetist before commencing the autopsy<sup>33</sup>. In some instances, the information yielded from the autopsy may be limited and careful correlation with the clinical findings is mandatory. Allergic reactions to anaesthetic agents are very uncommon and may be difficult to substantiate at autopsy since typical signs of anaphylaxis may not be apparent<sup>34</sup>. In addition, it is vital to insist that in any death where anaesthesia is an issue, the tubes, lines and other cannulae must be left in situ. The principal author has dealt with a case where the death was related to incorrect siting of an endotracheal tube. Since this had been removed (the patient had survived for some time after the incident with hypoxic brain damage), the only available evidence was documented in the notes with regard to the incorrect tube siting. Had the patient died whilst anaesthetised, the position of the tube may have been the only clue as to the real cause of the problem.

#### **Indirect Maternal Death**

#### **Cardiovascular Disease**

Cardiac disease is the commonest cause of indirect maternal death with 35 deaths recorded in the last enquiry. Cardiac disease includes both congenital<sup>35</sup> and acquired disease, the latter being the commoner<sup>2</sup>. In most cases, the presence of pre-existing heart disease will usually be appreciated, but with the presence of congenital disease comes the risk of endocarditis. This should be borne in mind in any pregnant woman presenting with non-specific symptoms. Endocarditis is now more common in women with an intravenous drug problem than in those with congenital heart disease<sup>36</sup>. Improvements in the treatment and management of women with congenital heart disease are such that there is no increase in mortality of these women in pregnancy, with the exception of Eisenmenger's Syndrome. However, the risk of heart failure is considerable and there is an increased risk of foetal loss and intrauterine growth retardation and premature labour and delivery<sup>37</sup>.

In acquired disease, problems such as aortic dissection (which may be associated with Marfan's Syndrome)<sup>38</sup>, cardiomyopathy and pulmonary hypertension occur. Myocardial infarction is uncommon and is usually associated with coronary artery atheroma or aortic dissection<sup>39</sup>. Puerperal (peripartum) cardiomyopathy accounted for seven deaths considered by the last enquiry and is uncommon<sup>40</sup>. In this condition, the heart becomes enlarged with left ventricular dysfunction<sup>41</sup> and may be viral in aetiology. There are no characteristic histological features, but using endocardial biopsy, there may be focal fibrosis, variability in myocyte calibre and scattered chronic inflammatory cells. Dilated cardiomyopathy is also recognised increasingly in association with HIV infection as this pandemic increasingly affects women (see below)<sup>42</sup>. Cardiomyopathy has also been observed in women with antiphospholipid syndrome<sup>43</sup>.

#### **Central Nervous System Disease**

These conditions account for a substantial number of indirect maternal deaths, most commonly due to epilepsy where there may be an increase in fit frequency during pregnancy and where anticonvulsant medication levels may be difficult to titrate. Subarachnoid haemorrhage accounted for a substantial number of deaths (eleven in the last enquiry). Occasionally, these patients are hypertensive, but not always so. Primary intracerebral haemorrhage caused nine deaths. Cerebral thrombosis is an uncommon cause of death but the morbidity is higher. In survivors of stroke, the aetiology may be related to eclampsia or, rarely, AFE, but the cause of the majority is undiagnosed and, therefore, a proportion of these strokes may actually be unrelated to the pregnancy<sup>21</sup>. In all cases, and particularly so in the case of subarachnoid haemorrhage, an autopsy is vital to establish the cause of death. A small but significant number of maternal deaths still do not have postmortem examinations performed. In a proportion of these, new information will be available which was not evident clinically during life.

#### **Other Causes of Indirect Death**

These include infectious disease; endocrine, metabolic and immunity disorders; disorders of the blood; circulatory disorders; diseases of the respiratory tract and

disorders of the gastrointestinal system; in total accounting for 75 deaths in the last enquiry. The whole spectrum of pathology can be seen in any woman of child-bearing age and for this reason, there is little excuse for an autopsy which is limited purely to look for "maternal problems".

# **Human Immunodeficiency Virus (HIV) in Pregnancy**

HIV infection is a world-wide problem which is expected to increase exponentially over the next few years<sup>44</sup>. As such, there is likely to be an increase in morbidity and mortality associated with HIV infection and more demand for specialist obstetric services geared toward women with this problem. In the UK, much of the infection was previously within the homosexual community, but heterosexual transmission is now an increasing dilemma. HIV is a particular problem in intravenous drug users in whom pregnancy presents a management challenge. The mortality of HIV-positive women does not appear to be increased in pregnancy, but HIV predisposes to several pregnancy-related problems including low birth weight, infection and prematurity<sup>45</sup>. With an increasingly mobile world population and with immigration from areas where the disease is endemic, HIV is now a major consideration in the management of a significant number of pregnancies. The virus is routinely screened for on a confidential basis at booking in the United Kingdom. Advances in anti-retroviral therapy have reduced the rate of transplacental transmission, but this may become less effective with the emergence of triple-drug-resistant strains.

With HIV infection, there are also problems of concomitant opportunistic infections. World-wide, the commonest of these is tuberculosis. Treatment of this infection can be effective with appropriate therapy, but again, drug resistance is bound to pose an increasing challenge.

HIV per se rarely precipitates maternal death, but opportunistic infections such as *Pneumocystis carinii* are a cause of significant mortality<sup>46</sup>. The maternal death autopsy on an HIV-positive woman, as with other bodies infected by HIV or other similar pathogens, poses a number of additional hazards to the pathologist<sup>47</sup>. As a rule, all autopsies in any case should be treated as potentially infectious, but the pathologist should take the usual recommended additional precautionary procedures. In addition, (s)he should also make sure that appropriate micro biological samples are retained. Examination of the placenta may yield useful information: there is an increased incidence of chorioamnionitis in HIV-positive women<sup>48</sup>.

#### **Deaths Related to Substance Abuse**

The management of pregnancy in the substance abuser is now becoming a specialised field in its own right<sup>49</sup>. Abuse of heroin is common and the use of cannabis is widespread, the latter rarely seeming to cause any problem. Pregnant heroin abusers are maintained on methadone programmes since abrupt cessation of opiates can be dangerous in itself. Death in pregnancy, either direct or indirect, in substance abusers is uncommon. Nevertheless, the pathologist should always be

aware that the mother may have had a drug problem and (s)he should take appropriate action in the autopsy room fully to investigate the death and also to protect against the potential hazards associated with high-risk autopsies.

#### **Late and Fortuitous Deaths**

In some instances, these deaths are related to problems occurring in the puerperium or advances in medicine prolonging the woman's survival. The majority of late deaths are related to indirect causes, usually neoplastic disease and cardiovascular problems.

Neoplastic disease of all types accounted for fifty-two deaths in the last enquiry with the reported incidence of 1 in 6,000 live births. Suicide is also uncommon but not unknown during pregnancy and the puerperium. Puerperal depression is common, but suicide is rarely completed, the presence of a baby seeming to confer a protective effect on the mother. Suicide can occur despite adequate psychiatric support and can sometime occur "out of the blue" where the woman was not previously considered to represent a suicide risk even thought receiving anti-depressant medication and support.

Domestic violence and homicide were included in the last enquiry in detail for the first time. The husband or partner is the usual perpetrator of the violence, with violence or death associated with those over 25 years of age and in the second trimester of pregnancy. Three of the eight reported cases of murder occurred in those of Indian or Pakistani ethnic origin.

Pregnant women may die as a result of road traffic incidents. This may be whilst drivers or passengers within motor vehicles or when they are external to a vehicle, for example as a pedestrian, spectators at car rallies or pillion passengers on motorbikes. For those within vehicles, the correct type of seat belt must be worn in the correct position. Examples of how to and how not to wear a seat belt whilst pregnant can be found within the last confidential enquiry<sup>2</sup>.

Finally, particularly in developing countries, a number of deaths occur due to burns. In these cases, morbidity and mortality are high especially if in the third trimester.

It is likely that the number of fortuitous deaths and late death is under-recorded. This is probably due to the doctor who is reporting the death failing to record the fact that the woman was recently pregnant.

# The Autopsy

All pathologists who wish to embark upon a maternal autopsy are recommended to review the report of Rushton and Dawson<sup>50</sup>. One must also read all maternal medical records to fully understand the past medical history and ante-natal care. Consideration should also be given to talking to the obstetrician prior to the autopsy and if possible the obstetrician should be encouraged to attend the examination. Thus, successful investigation depends upon consideration of the potential problems in the assessment of the notes and relevant drug history before starting the examination itself.

#### **External Examination**

The external examination should be no different from any other autopsy examination in the fact that it should record the physical and ethnic attributes of the deceased and document all natural disease, marks of medical intervention, old and fresh changes or marks of violence. Particular attention should be directed to features relevant to possible problems encountered in maternal death and the external findings should be correlated with the clinical notes.

Cannulae and other paraphernalia of medical intervention should be left in situ in the body. The presence of surgical scars should be noted and documented. This assumes importance in examples of uterine rupture where there has been a previous caesarean section. Other scars may indicate previous surgery for congenital heart disease.

The legs should be examined carefully. Swelling of a calf may indicate the presence of a deep vein thrombosis (DVT) although the absence of obvious swelling does not preclude the presence of a DVT. Generalised oedema may be a reflection of pre-eclampsia or exacerbation of congenital heart disease. Attention should be paid to the external genitalia, particularly if there has been instrumentation during delivery, e.g., forceps or ventouse extraction. In the presence of a vaginal discharge, appropriate microbiological swabs are mandatory.

As domestic violence is an increasingly recognised cause of maternal death, all injures should be documented. This will also include marks of self-harm or illicit drug use.

#### Internal Examination

The post mortem examination should, of course, be complete with regard to coroner's examinations and all systems examined. In the case of the "consent" autopsy, the examination may have to be limited to those areas for which consent to examination has been given. In this case, the pathologist may have to advise the relatives, with the assistance of clinicians, as to what is likely to be required in order to perform an adequate examination. Given the current difficulties regarding obtaining relatives' consent for autopsy and the confusing nature of some consent forms, it is not uncommon for relatives to consent to a limited examination, only for the pathologist to find that an area vital to the examination cannot be inspected because of that limited consent. In such cases, the examination effectively becomes useless.

The maternal death autopsy does not differ from any other post mortem examination. However, modification of one's usual technique may be required to demonstrate some relevant pathologies. Successful detection of many of the problems of the pregnancy-related death depends to a large extent upon the pathologist considering the potential pathology before opening the body. Some particular problems have been highlighted in the preceding text and dissection techniques in order to demonstrate some of these problems are given below.

### Pulmonary Thromboembolism

The pulmonary trunk should ideally be examined before the lungs are detached from the thoracic pluck. The pathologist is strongly recommended to do the dissection himself since removal of the lungs, even by an experienced technician, may

result in loss of thromboembolus from the pulmonary trunk. Once the pulmonary trunk has been inspected the remainder of the pulmonary arterial tree can be examined, as previously described by McCulloch and Rutty<sup>51</sup>.

#### Air Embolism

This can be very difficult to demonstrate, but several possible techniques can be attempted although none are ideal. Not infrequently, a slight frothiness can be seen in the blood within the pulmonary trunk where there is no question of air embolism, but large air emboli are likely to cause a substantial quantity of froth in this vessel.

Before opening the neck, the abdominal organs should be carefully displaced on opening the abdominal cavity. In large air emboli, gas bubbles may be observed within the inferior vena cava, but given the fact that this vessel usually collapses after death, diagnosing air embolism in this site may be very difficult. Older texts advocate examining the cerebral veins for air bubbles, but this is probably of no value as the majority of these so-called "cerebral air emboli" are in fact post-mortem artefacts.

Air embolism should be ideally demonstrated in the heart. Prior to the autopsy a chest x-ray or preferably a computerised tomography scan (CT Scan) should be performed, which may show the characteristic appearance of air within the right side of the heart or great vessels draining into the right atrium. The anterior chest wall should be removed. There are two techniques described to demonstrate air within the heart<sup>52</sup>. The easiest of these techniques is when the pericardial sac is carefully opened on the anterior surface and then filled with water. Small incisions are made in the right atrium and ventricle beneath the water line. Where there has been substantial air embolism, bubbles should flow into the water from the heart.

#### Genital Tract Trauma

Adequate demonstration of structural and traumatic problems in the genital tract is most easily demonstrated by en bloc dissection of the internal and external genitalia. This involves removal of the perineum in direct continuity with the vagina, uterus and adnexae. This technique requires practice and most general pathologists will not have had reason to attempt the dissection in routine practice. A specialist dissection manual should be consulted<sup>53</sup>. If in doubt, advice may be sought from a forensic pathologist, most of whom use the technique properly to examine genital injuries in cases of violent sexual assault.

Using en bloc dissection allows orderly inspection of the perineum, vagina, cervical os and uterine wall. The en bloc technique requires additional reconstruction after the organs have been removed. For this reason, and given the current climate of public concern regarding autopsy practice, the pathologist is advised to warn the coroner if this type of dissection is required. In the "consent" autopsy, the necessity for this dissection will require sensitive discussion with relatives.

#### Haemorrhagic Disorders of Pregnancy

As indicated above, the commonest problems are placenta praevia, placental abruption and post-partum haemorrhage. Placental abruption can be remarkably difficult to diagnose, particularly if the placenta has been delivered and is not available for inspection. Even if the placenta has been retained, the blood clot

may have become detached. The endometrium should be inspected for the site of placental implantation and any retained products should be noted. Appropriate histological material should be retained to demonstrate placenta accreta.

#### **Hypertensive Disorders**

There may be evidence of intracranial haemorrhage, liver capsule haematoma or other stigmata of a haemostatic problem. There is an overlap in the clinical signs of HELLP syndrome and acute fatty liver of pregnancy (AFLP). The latter is usually obvious macroscopically in the form of a yellow greasy liver and microscopically, fat being demonstrable using a frozen section/Oil Red O technique.

#### **Anaesthetic Problems**

Demonstrating incorrectly placed catheters, lines and tubes is less of a problem providing all medical impedimenta are left in situ. Aspiration of gastric contents can occur if a cuffed endotracheal tube is not used. This may be obvious on macroscopic examination of the cut surfaces of the lungs, but in any event, consideration should be given to retaining histological samples, since aspiration is easier to demonstrate microscopically. Inappropriate administration of anaesthetic agents and gases may be almost impossible to demonstrate at autopsy and the diagnosis is largely made clinically. Allergic reactions to anaesthetic agents are unusual and there may be few histological features of this. Measurement of serum tryptase levels may be of assistance<sup>34</sup>. The advice of the local chemical pathologist or immunologist may be helpful.

# **Retention of Samples**

These conform to the usual categories: histology, microbiology, biochemistry and neuropathology, with additional consideration to toxicology, immunology, genetic and metabolic studies. Any samples taken should be recorded in the autopsy report, even if the investigation is subsequently found to be negative. Both the coroner and the relatives must be informed of exactly what has been retained so that the option of returning the material to the body prior to disposal of the body is fully discussed. The use of a pro forma such as shown in Fig 4.1 will assist this process.

# Histology

All organs should be sampled where possible. In addition, particular consideration should be given to those items listed in Table 4.3, which is a suggestion only: sampling will need to be tailored to individual cases.

Histochemistry and immunohistochemistry stains may be essential. Consideration should be given to the following:

- Demonstration of disseminated intravascular coagulation (DIC): Martius Scarlet Blue for fibrin can be used on blocks of kidney and lung (Fig 4.3).
- Pulmonary hypertension: Elastic stains on sections of lung to demonstrate vasculature.
- Amniotic fluid embolism: A modified phloxine tartrazine technique (Attwood's technique) is recommended although a broad-spectrum cytokeratin stain may

Table 4.3	Suggested	l areas of	f sampli	ing for	histo	logical	examination.
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Cardiovascular system Respiratory system	Both ventricles, septum and atria. One block from each lobe and blocks to include intermediate bronchi: the latter are useful for demonstrating aspiration and acute asthmatic attack. Trachea and epiglottis.
Gastrointestinal system	Liver, pancreas and any intestinal lesion. The latter two tend to degenerate rapidly and histology may actually be of little practical use. Liver should be frozen for fat examination.
Internal genitalia	Placental bed and full-thickness uterine wall, cervix, ovaries, fallopian tubes and any vaginal lesion. The broad ligament to include the veins should be sampled.
Urinary system	Both kidneys, bladder (in cases of trauma).
Central nervous system	Motor cortex, hippocampus, brainstem, cerebellum and basal ganglia

achieve the same effect. The former may not be available in some laboratories, so the pathologist may have to do some local research if this technique is required.

 A simple frozen section/Oil Red O stain is often useful in the demonstration of fat in pulmonary capillaries and in the liver.

#### **Chemical Pathology/Immunology**

The types of samples which may be required have been discussed above although these should include peripheral blood, urine, and stomach contents. Vitreous can be taken for electrolyte and glucose assessment. If necessary, specialist advice should be sought before the autopsy commences.

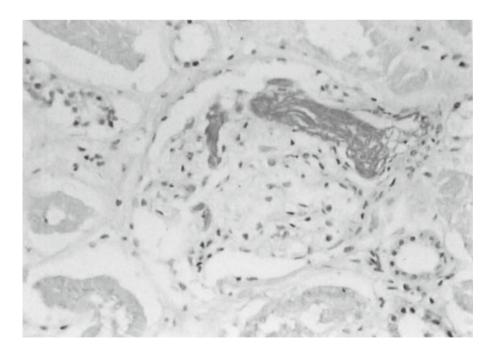


Fig. 4.3 Amniotic fluid embolism within the kidney. Phloxine tartrazine.

#### Microbiology

Swabs of the genital tract are required to demonstrate sepsis. Those from the perineum and vagina should be taken before dissection commences. The samples should, of course, be appropriately labelled and the laboratory advised that the patient was pregnant and also of any antibiotics which were administered to the patient. Sepsis may not be suspected ante mortem and the pathologist is advised to take appropriate samples before dissection and then to discard them if they are later found to be unnecessary. These would include blood for culture, samples of lung and spleen for bacteriology and virology as well as cerebrospinal fluid either by aspiration or swab.

#### Neuropathology

Occasionally, it may be necessary to retain the brain, for example, in deaths related to epilepsy, fits due to eclampsia or acute Wernicke's encephalopathy. This may happen if there is suggestion of hypoxic brain damage following collapse or if there has been an anaesthetic problem. Retention of this, or indeed any organ, must be handled very sensitively. In cases of cerebral pathology, the ideal is, of course, to fix the brain and then examine it properly, but the necessary consent for this may be lacking. A reasonable compromise may be to retain adequate samples of appropriate areas whist not retaining the whole brain<sup>54</sup>. However, the relatives should be aware that not examining the brain where the findings may be relevant to the death may subsequently limit the usefulness of the examination in determining the cause of death or regarding the pursuit of any subsequent civil claim for medical negligence. The pathologist must ultimately be guided by the coroner or consent of the relatives, and the pathologist must provide advice for any consent to be appropriately informed.

#### **Examination of the Placenta and Foetus**

If retained, examination may reveal features of pre-eclampsia in the form of atherosis of the arterioles, infarction and fibrin deposition, features of chorioamnionitis and, rarely, tumours. Abruption may be difficult to assess if the clot has become detached from the placenta. In cases of intra-uterine or peri- or postpartum death, examination of the foetus is also of primary importance. It is entirely appropriate to seek the specialist opinion of a paediatric or gynaecological pathologist in the assessment of specialist material.

# The Autopsy Report

In this, the pathologist should aim to document as many findings as possible, even if they are negative. The Confidential Enquiry highlights poor pathology reports as a common problem (see above): this is often as a consequence of inadequate histological examination or insufficient clinico-pathological correlation. The pathology report may form the basis for evaluation of the case by the Local Assessor and the report should attempt specifically to address any clinical problems which were encountered. As with any autopsy report, the pathologist should bear in mind that

it may not necessarily always be a medically qualified person who is reading it and, therefore, medical terminology should be appropriately explained for the lay reader. The pathologist should also remember that one person who may read the report is a solicitor. In any event, the pathologist should make the report as full as possible since this will be the most useful aide memoire at the time of any inquest (s)he may be required to attend.

# **Acknowledgements**

We are grateful for the technical support of Professor J. Crane and Mr Sam Nelson, and also for the comments of Dr J.H.F. Smith.

#### References

- 1. Ibison JM, Swerdlow AJ, Head JA, Marmot M. Maternal mortality in England and Wales 1970-1985: An analysis by country of birth. Br J Obstet Gynaecol 1996;103:973-80.
- 2. Department of Health. Why mothers die. The Fifth report of Confidential Enquiries into Maternal Deaths in the United Kingdom 1997-1999. The Royal College of Obstetricians and Gynaecologists Press: 104.
- 3. Ahmed Y, Mwaba P, Chintu C, Grange JM, Usianowski A, Zumula A. A study of maternal mortality at the University Teaching Hospital, Lusaka, Zambia: The emergence of tuberculosis as a major non-obstetric cause of maternal death. Int J Tuberc Lung Dis 1999;3:675-80.
- 4. Keeling JW, Gray ES. Maternal death. In: Payne-James J, Busuttil A, Smock W, editors. Forensic medicine: Clinical and pathological aspects. London: GMM, 2002;213-30.
- 5. Abortion Act, 1967.
- 6. De Swiet M. Thromboembolism in pregnancy. In: Weatherall DJ, Ledingham JGG, Warrell DA, editors. Oxford textbook of medicine, 3rd ed. Oxford: Oxford University Press, 1996;1741-4.
- 7. Yerby MS. Contraception, pregnancy and lactation in women with epilepsy. Baillieres Clin Neurol 1996;5:887-908.
- 8. Crawford P. CPD Education and self-assessment: Epilepsy and pregnancy. Seizure 2001;
- 9. Burton J, Rutty G. The hospital autopsy 2nd ed. London: Arnold, 2001.
- 10. Coroners' Act, 1988.11. Coroners' Rules, 1984.
- 12. Human Tissue Act, 1961.
- 13. Dorries C. Coroner's Courts: a guide to law and practice. Wiley: London, 1999.
- 14. The Royal Liverpool Children's Enquiry. London: The Stationery Office. 2001.
- 15. Royal College of Pathologists. Guidelines for the retention of tissues and organs at post-mortem examination. London: Royal College of Pathologists, 2000.
- 16. Rai R, Regan L. Antiphospholipid syndrome and pregnancy loss. Hosp Med (London) 1998; 59:637-9.
- 17. Amengual O, Atsumi T, Khamashta MA, Hughes GR. Advances in antiphospholipid (Hughes') syndrome. Ann Acad Med Singapore 1998;27:61-6.
- 18. Cowchock S. Treatment of antiphospholipid syndrome in pregnancy. Lupus 1998; 7 Suppl 2: S95-7.
- 19. Bates SM. Optimal management of pregnant women with acute venous thromboembolism. Haemostasis 1999; 29 Suppl S1:107-11.
- 20. Lockwood CJ. Heritable coagulopathies in pregnancy. Obstet Gynaecol Surv 1999;54:754-65.
- 21. Simolke GA, Cox SM, Cunnigham FG. Cerebrovascular accidents complicating pregnancy and the puerperium. Obstet Gynaecol 1991;78:37-42.
- 22. Mas JL, Lamy C. Stroke in pregnancy and the puerperium. J Neurol 1992;245:305-13.
- 23. Leitch CR, Cameron AD, Walker JJ. The changing pattern of eclampsia over a 60-year period. Br J Obstet Gynaecol 1997;104:917-22.
- 24. Thomson AJ, Greer IA. Non-haemorrhagic obstetric shock. Baillieres Best Pract Res Clin Obstet Gynaecol 2000;14:19-41.
- 25. Khong TY. Expression of endothelin-1 in amniotic fluid embolism and possible pathophysiological mechanism. Br J Obstet Gynaecol 1998;105:802-4.

- 26. Fletcher SJ, Parr MJ. Amniotic fluid embolism; a case report and review. Resuscitation 2000;43:141-6.
- 27. Fineschi V, Gambassi R, Gherardi M, Turillazzi E. The diagnosis of amniotic fluid embolism: an immunohistochemical study for the quantification of pulmonary mast cell tryptase. Int J Legal Med 1998;111:238–43.
- 28. Oi H, Kobayashi H, Hirashima Y, Yamakazi T, Kobayashi T, Terao T. Serological and immunohistochemical diagnosis of amniotic fluid embolism. Seminars in Thromb Haemost 1998;24:479-84.
- 29. Attwood HD. The histological diagnosis of amniotic fluid embolism. J Pathol Bacteriol 1958;76:211–15.
- 30. Garland IWC, Thompson WD. Diagnosis of amniotic fluid embolism using an antiserum to human keratin. J Clin Pathol 1983;36:625-7.
- 31. Jones H, Diop N, Askew I, Kabore I. Female genital cutting practices in Burkina Faso and Mali and their negative health outcomes. Stud Fam Plann 1999;30(3):219-30.
- 32. McCaffrey M, Jankowska A, Gordon H. Management of female genital mutilation: the Northwick Park Hospital experience. Br J Obstet Gynaecol 1995;102(10):787-90.
- 33. Start RD, Cross SS. ACP Best Practice No. 155. Pathological investigation of deaths following surgery, anaesthesia and medical procedures. J Clin Pathol 1999;52:640-52.
- 34. Pumphrey RS, Roberts IS. Post-mortem findings in fatal anaphylactic reactions. J Clin Pathol 2000;53:273–6.
- 35. Sawhney H, Suri V, Vasishta K, Gupta N, Devi K, Grover A. Pregnancy and congenital heart disease maternal and fetal outcome. Aust NZ J Obstet Gynaecol 1998;38:266–71.
- 36. Cox SM, Leveno KJ. Pregnancy complicated by bacterial endocarditis. Clin Obstet Gynaecol 1989;32:48-53.
- 37. Schmaltz AA, Neudorf U, Winkler UH. Outcome of pregnancy in women with congenital heart disease. Cardiol Young 1999;9:88–96.
- 38. Pyeritz RE. Maternal and fetal complications of pregnancy in the Marfan's syndrome. Am J Med 1981;71:784–90.
- 39. Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. Ann Int Med 1996;125:751-62.
- 40. Heider Al, Kuller JA, Strauss RA, Wells SR. Peripartum cardiomyopathy: a review of the literature. Obstet Gynaecol Surv 1999;526–31.
- 41. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, Ansari, Baughman KL. Peripartum cardiomyopathy. National Heart, Lung and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. J Am Med Assoc 2000;283:1183–8.
- 42. Cetta F, Michels VV. The natural history and spectrum of idiopathic dilated cardiomyopathy, including HIV and peripartum cardiomyopathy. Curr Op Cardiol 1995;10:332–8.
- 43. Airoldi ML, Eid O, Tosetto C, Meroni PL. Post-partum dilated cardiomyopathy in antiphospholipid positive woman. Lupus 1996;5:247-50.
- 44. MacGregor SN. Human immunodeficiency virus infection in pregnancy. Clin Perinatol 1991; 18:33–50.
- 45. Landers DV, Martinez de Tejada B, Coyne BA. Immunology of HIV and pregnancy. The effects of each on the other. Obstet Gynaecol Clin N Am 1997;24:821-31.
- 46. Hicks ML, Nolan GH, Maxwell SL, Mickle C. Acquired immunodeficiency syndrome and *Pneumocystis carinii* infection in a pregnant woman. Obstet Gynaecol 190;76:480-1.
- 47. Claydon SM. The high-risk autopsy. Recognition and protection. Am J Forensic Med Pathol 1993;14:253-6.
- 48. Chadwani S, Greco MA, Mittal K, Antione C, Krasinski K, Borkowsky W. Pathology and human immunodeficiency virus expression in placentas of seropositive women. J Infect Dis 1991; 163:1134–8.
- 49. Silver H, Wapner R, Loriz-Vega M, Finegan LP. Addiction in pregnancy: high-risk intrapartum management and outcome. J Perinatol 1987;7:178–84.
- 50. Rushton DI, Dawson IMP. The maternal autopsy. J Clin Pathol 1982;35:909-21.
- 51. McCulloch TA, Rutty GN. Post-mortem examination of the lungs: a preservation technique for opening the bronchi and pulmonary arteries individually without transection problems. J Clin Pathol 1998;51:163–6.
- 52. Rutty GN, Burton JL. Demonstration of a cardiac air embolus. In: Burton JL, Rutty GN, editors. The hospital autopsy, 2nd ed. London: Arnold, 2001;85–6.
- 53. Rutty GN, Burton JL. Examination of the female genital tract. In: Burton JL, Rutty GN, editors. The hospital autopsy, 2nd ed. London: Arnold, 2001;91–3.
- 54. Timperley WR. ACP Best Practice No.158. Neuropathology. J Clin Pathol 2000;53:255-65.

# 5. The Pathology of Shock Versus Post-mortem Change

G.N. Rutty

#### Introduction

It does not matter what type of autopsy-based pathology you practice, be it foetal, neonatal and paediatric, adult, neuropathological or forensic, you will come across many cases where the patient has suffered localised or systemic organ failure and "shock" as part of their terminal illness. This may be suggested within the clinical history or documented within the hospital notes but needs to be confirmed during the autopsy examination. However, if there is a delay between death and the autopsy examination then post-mortem autolysis will occur which may, especially if the time period goes beyond a day, confuse the picture presented to the pathologist. It is with this in mind that this chapter does not discuss the physiology of shock but rather concentrates on the macroscopic and microscopic pathology of shock and how, when possible, to differentiate the changes from that of postmortem autolysis. Although the pathological descriptions of shock have been documented previously within the medical literature, these texts have, at times, been poorly illustrated. This chapter brings these texts together and concentrates on illustrating the macroscopic and microscopic changes of shock to provide a comprehensive reference source on this subject.

# What do we mean by "Shock"?

When requested to fill in a death certificate, many autopsy-based pathologists rightly due not use the unqualified word "shock". This is simply because the word may be misinterpreted to mean one of many things. An example of this is given within the Concise Oxford Dictionary<sup>1</sup>, where the word "shock" is defined as:

- A violent collision, impact, tremor, etc.,
- A sudden and disturbing effect on the emotions, physical reactions, etc.,
- An acute state of prostration following a wound, pain, etc.,
- Electric shock,
- A disturbance causing instability in an organisation, monetary system, etc.,

- A group of usually 12 corn-sheaves stood up with their heads together in a field,
- An unkempt or shaggy mass of hair.

The word shock is derived from the Dutch word *schokken*, meaning to jolt or jerk suddenly and as can be seen from this non-exhaustive list, has many meanings depending upon the context and person to whom it is conveyed. One of the best examples of the many meanings of the word shock is found within the memoirs of Lord Moran. In 1914 a regimental medical officer in the British Army examined a sergeant who appeared "morose" and "apathetic". He recalled:

I found him staring into the fire. He had not shaved and his trousers were half open. I could get nothing from him...he did not appear to be ill. We agreed to let him rest, to let him stay in his billet till the battalion came out of the trenches. But next day when everyone had gone up the line he blew his head off. I thought little of it at the time, it seemed a silly thing to do.

This man was suffering from shock – shell shock; the functional disorder of the nervous system caused by an explosion of a large shell<sup>2</sup>. It is this illustration of a meaning of the word shock, especially associated with the concept of fear, that members of the general public understand as the meaning of the word shock rather than the medical condition. It is for this reason that doctors should qualify the word before using it.

The clinical use of the word shock has been used for several centuries. Pare (1510–1590) described the clinical state of cold sweat, pallor and absence of a detectable pulse although it was not until 1741 that Le Dran described what we now recognise as surgical or circulatory shock on page 6 of the book *Traite ou Reflectons tirees de la Pratique sur les Playes d'Armes a feu*<sup>3</sup>.

In pathological terms there are four causes of shock, where shock is defined as an inadequate perfusion of the tissues (referred simply to as shock from this point onwards within the text)<sup>4</sup>:

- 1. Cardiac shock: due to acute myocardial failure; for example, due to the infarction of the left ventricle or the infection i.e. a myocarditis.
- 2. **Hypovolaemic shock:** when there is a significant reduction in blood volume; for example, due to blood loss, burns or dehydration.
- Septic shock: when there is a gram positive or negative bacterial infection, fungi
  or opportunistic infection that may be associated with the liberation of endotoxins.
- 4. Anaphylactic shock: resulting in the degranulation of mast cells following binding of IgE; for example, to components of food, drugs or contrast medium.

It is the resulting hypoperfusion of the tissues that leads to hypoxia of the tissues and ischaemia. The number and range of organs involved in any single case is in turn dependent upon the sensitivity of the tissue to ischaemia and the duration of the hypoperfusion. The worst-case scenario will be multiorgan failure and death.

This chapter will consider those organs of the body in which pathological changes can be observed in the above four types of shock. In each case the changes that occur due to post-mortem autolysis will be considered and advice given on how, when possible, to differentiate this from the changes of ante-mortem shock.

# The Lungs

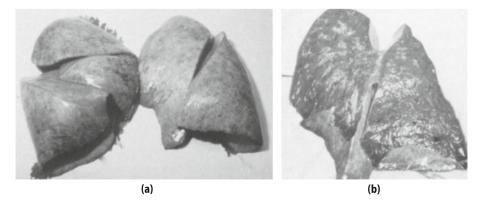
The lung is the commonest organ to be affected by shock. Unlike some of the other systems of the body it can be affected, to a lesser or greater degree, by all of the causes listed above. Pathology may be seen in up to 50.3% of those with hypovolaemic shock and 65% suffering from septic shock whereas in cardiogenic shock, lesions are less frequent, as the patient may die too quickly from the cardiac event for lung pathology to develop.

The earliest accounts of the effect of shock on the lungs were first described in World War II war casualties<sup>5</sup> and since then have been described under a multitude of names including "shock lung", "traumatic wet lung", "post-traumatic pulmonary insufficiency" and "Da Nang lung". The modern-day term for the appearances is Adult Respiratory Distress Syndrome (ARDS)<sup>7</sup>. The diagnosis is likely to have already been made during life as the patient will have developed severe dyspnoea, hypoxaemia, failing pulmonary compliance and bilateral diffuse pulmonary infiltrates on chest x-ray.

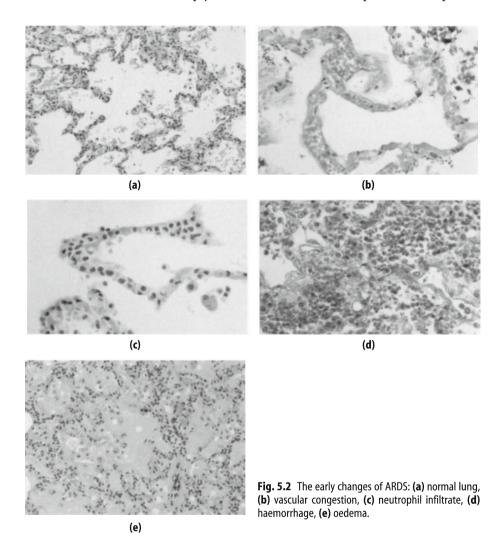
By the time that the pathologist is required to examine the lungs the changes may be in an advanced stage. The classical macroscopic appearances of the lungs in ARDS are those of bilaterally, uniformly enlarged, solid, airless lungs. The normal lobar architecture is over pronounced. The weight is increased, classically over 1 kg each. The cut surface of each lung is dry and petechial haemorrhages may be seen to the pleural and cut surfaces (Fig. 5.1).

The first microscopic changes seen in the lungs occur within 18 hours of the shock event and progressively become more pronounced between the next 18 to 72 hours. Ultimately, this first phase can last from 2 to 6 days. These changes are due to the effect on the endothelial cells lining the alveolar capillaries which are damaged due to the initial insult<sup>8,9</sup>.

This damage and generalised vascular vasodilation leads to the earliest changes, which can be summarised as follows: congestion of the vessels, polymorph sequestration, haemorrhage into the alveoli and pulmonary oedema (Fig. 5.2). Within this time period fibrin and platelet aggregates and thrombi may be found within the alveoli capillaries (Fig. 5.3). These, where present, do not result in complete occlusion of the vessel lumen. The necrotic type 1 pneumocytes slough off the

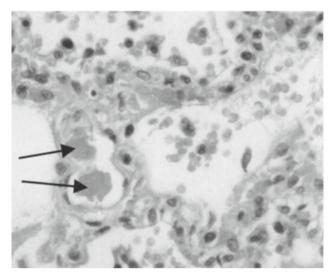


**Fig. 5.1** (a) Macroscopic appearance of the lungs in a case of ARDS. The lobar architecture is pronounced. (b) Cut surface of the left lung in ARDS. The lobes have a solid airless appearance.



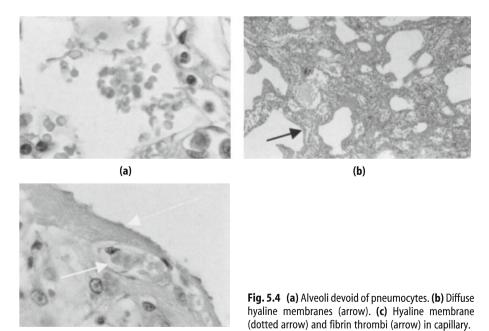
alveoli and go into the airspaces along with the fluid and proteins liberated from the capillaries due to the endothelial cell damage<sup>10</sup>. These, along with fibrin, are precipitated to form the so-called "hyaline membranes" which line the alveoli spaces (Fig. 5.4). These have been observed to start first on the tips of the alveolar walls along the ducts, gradually increasing in size to appear to outline the alveolar ducts and seal of the alveolar sacs<sup>11</sup>. They may reach up to 100 microns thick.

The last change that occurs in this acute stage of ARDS is a generalised increase in the number of megakaryocytes within the lung vessels. The normal population of megakaryocytes within the lung is estimated at 4–40 mk/cm² (depending upon the publication). However, in a number of pathological processes including shock the number increases to 60–1,200 mk/cm² (Fig. 5.5). This is thought to be due to an increase in release of megakaryocytes from the bone marrow as a result of stimulation of megakaryocyte-thrombocytopoiesis following the consumption of platelets during consumption coagulopathy¹².

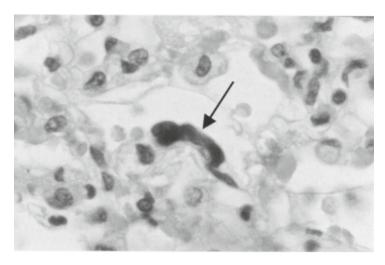


**Fig. 5.3** Fibrin thrombi within the lung capillaries (arrows). Note cellular congestion of capillaries with haemorrhage within the alveoli.

Overlapping the first stage of ARDS in the time sequence is the start of the recovery phase. This may occur as early as 48 hours, or more commonly is delayed for up to 72 hours after the onset. During this phase the principal changes seen to the lung are: proliferation of type 2 pneumocytes, infiltration by macrophages/plasma cells, myofibroblast proliferation and new basement membrane formation.



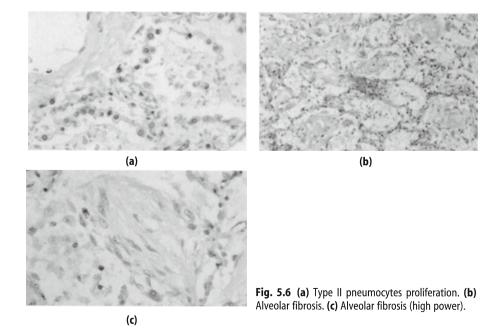
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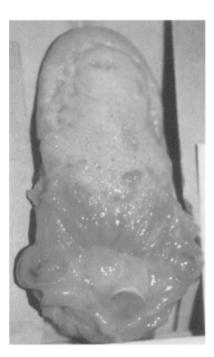


**Fig. 5.5** Lung megakaryocyte (arrow) numbers are increased in ARDS.

The final event in the sequence is interstitial and intra-alveolar organisation and fibrosis which typically occurs by 8 days (Fig. 5.6). This may lead to death within a period of 3 weeks as the alveoli are obliterated by fibrosis.

As with other organs of the body, the respiratory system may show minimal if any changes in those dying from anaphylactic shock due to the rapid time period between the onset of symptoms and death. Having said this, changes may be seen





**Fig. 5.7** Oedema of the epiglottis of a person dying from anaphylactic shock following exposure to a food allergen.

to the system but these may be confused with other lung pathology, which often co-exits in patients liable to anaphylactic reactions. The principal differential diagnosis is that of asthma as the macroscopic changes are those of hyper-inflated lungs, air trapping and bronchial mucus plugging. The microscopic picture is that of acute asthma. The pathologist must consider an anaphylactic event and undertake appropriate immunological investigations<sup>13</sup>. In the case of anaphylaxis caused by food allergens, upper airway obstruction may be seen due to the development of angiooedema. This may occur at the point of contact of the allergen with the tissues; for example, the lips or the tongue, which may swell within minutes of exposure to the allergen. Angio-oedema of the neck with oedema of the hypopharynx and epiglottis may result in respiratory obstruction and death (Fig. 5.7).

The differentiation between ante-mortem shock and post-mortem autolysis in the lungs may be extremely difficult if death has occurred prior to the development of hyaline membranes. Once this stage has occurred or the lungs have advanced into the healing stages then these changes should be used to differentiate between the two. In the post-mortem period as the lungs undergo autolysis they retain their connective tissue framework and vascular walls for several weeks.

The bronchial cartilage can be identified as well as any anthrocotic pigment that may be present. However, the alveoli may contain accumulated fluid, blood and cell-like structures which have an appearance similar to the acute changes of shock (Fig. 5.8). Although some authors have tried to distinguish ante-mortem, protein-rich fluid from post-mortem decomposition, it is considered unreliable to undertake such work and therefore at the end of the day one may not be able to distinguish between the two changes<sup>4</sup>.

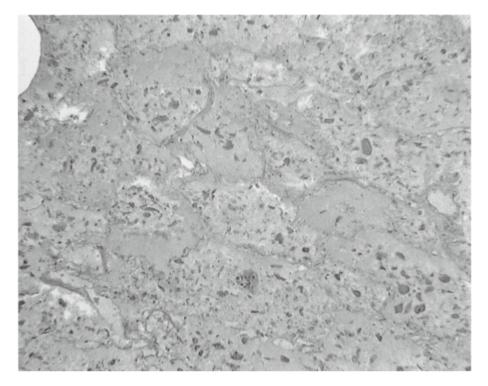


Fig. 5.8 Post-mortem autolysis of the lung.

### The Heart

The heart is the second commonest organ within which pathological changes may be observed in shock. These may be seen in 55.7% of cases of cardiogenic shock, 37.3% of hypovolaemic shock and 17% of septic shock<sup>14–18</sup>. In the case of cardiogenic shock, the resulting consequences of shock may lead to reduced perfusion of the left ventricle and secondary myocardial necrosis. This in turn will result in further cardiogenic shock. This cycle of shock-necrosis-shock may result in the deterioration of the patient and death. At autopsy it may then become difficult to distinguish between the triggering event and the complications of cardiogenic shock<sup>19</sup>.

Historically, the changes that we now associate in the heart with haemorrhagic shock were first reported by Wiggers in 1950 who developed a method of producing haemorrhagic shock in dogs<sup>20</sup>. Later authors identified that there was a point up to which the hypotensive state was reversible (sublethal bleeding volume), beyond which the degree of blood loss became irreversible (lethal bleeding volume)<sup>21–23</sup>.

The principal features of shock in the heart are those of haemorrhage and necrosis. All age groups can be affected although those with predisposing disease – for example, coronary artery atheroma – will be more susceptible to the effects of shock. The lesions can be found distributed throughout the heart either as single-fibre



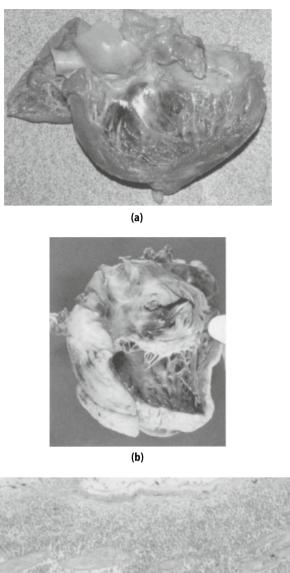
Fig. 5.9 Petechial haemorrhages are seen along the course of the coronary arteries.

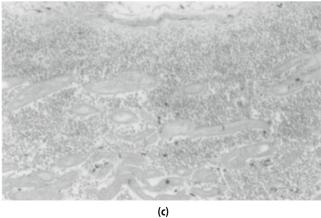
involvement or extensive areas of infarction. The conducting system is not uncommonly affected especially in fatal cases.

Macroscopically, the heart may show no findings at all although areas of haemorrhage may occur both externally and internally. To the surface of the heart, petechial haemorrhages may be seen along the courses of the coronary arteries (Fig. 5.9). Internally, the commonest finding is that of subendocardial haemorrhage to the left ventricular outflow tract (Fig. 5.10 (a)). Haemorrhage may also be seen to the right side of the heart, in both the atrium and ventricle, although these as less frequent than to the left ventricle (Fig. 5.10 (b)). This haemorrhage can develop extremely quickly and may be seen to those dying of haemorrhagic shock; for example, due to overwhelming blood loss from a fatal stab wound, where the time between the infliction of the wound, loss of consciousness and death is only a matter of minutes. This haemorrhage can be confirmed to be within the subendocardial area by microscopy (Fig. 5.10 (c)).

Unlike the distribution of myocyte necrosis seen, for example, in a regional myocardial infarct due to coronary artery disease, necrosis is commonly subendocardial due to inadequate myocardial perfusion, and ranges from microscopic foci to full-thickness transcircumferential lesions. A spectrum of changes can be seen ranging from contraction band necrosis to myocytolysis to coagulative necrosis<sup>24–27</sup>. By themselves these changes are not specific for shock as they can be seen due to other pathologies; for example, hyperkalaemia or in the presence of high circulating catecholamines.

Contraction band necrosis (formerly known as tigroid banding) may be seen in the subendocardial region of the heart or in the vicinity of coagulative necrosis. Clusters of myocytes are seen to contain deeply eosinophilic staining bands. These bands represent the telescoping of the sarcomeres and are thought to occur due to an element of reperfusion (Fig. 5.11). The features of coagulative necrosis are described within other standard texts of cardiovascular pathology with the macroscopic and microscopic features being time dependent. Another reported change





**Fig. 5.10** (a) Haemorrhage to left ventricular outflow tract. (b) Haemorrhage to right atrium in area of the AV node. (c) Endocardial haemorrhage.

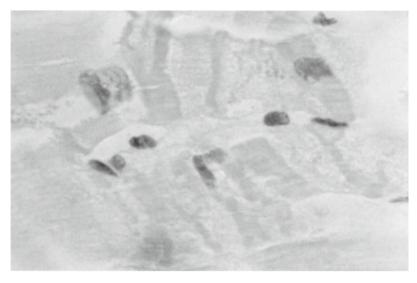


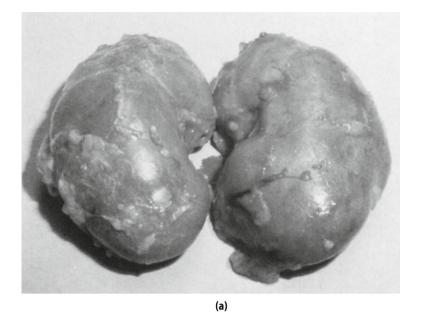
Fig. 5.11 Contraction band necrosis.

in the heart in shock is the appearance of wavy, attenuated fibres with the loss of nuclei.

A number of authors have undertaken histochemical and electron-microscopic studies on the changes to the heart muscle after death, where in the early post-mortem period heart muscle is considered to be relatively resistant to post-mortem autolysis<sup>4</sup>. Haemorrhage, contraction bands and focal areas of necrosis with inflammatory cell infiltrates are not features of cardiac post-mortem change. Ultimately,



**Fig. 5.12** Autolytic myocytes showing ghost structures lacking cellular detail, haemorrhage, contraction bands, inflammation or wavy shape.



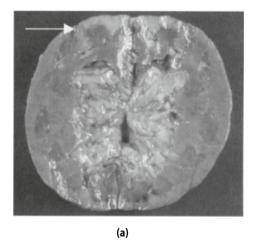


**Fig. 5.13** (a) The kidneys in shock. They are swollen with tense capsules. (b) The cut surface shows loss of normal architecture.

the microscopic examination of post-mortem heart will show eosinophilic ghost myocytes (Fig. 5.12).

# The Kidneys

Unlike, for example, the lungs, although renal function may be severely compromised in shock, the correlation between clinical symptoms and renal pathology is often poor. Changes may be seen to the components of the kidney, and are confined to the cortex in 24.5% of cases of hypovolaemic shock, 11.2% of cardiogenic shock and 18% of those with septic shock. Although the principle underlying pathology is that of acute tubular necrosis, the other components of the cortex – i.e., the glomerulus, the vessels and the interstitial connective tissue – may all show changes to a lesser or greater degree.



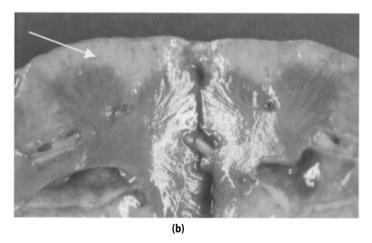
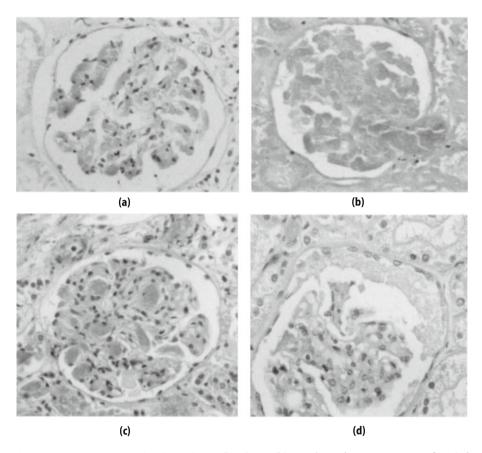


Fig. 5.14 (a) Area of cortical necrosis (arrow). (b) Magnified view of area of cortical necrosis (arrow).

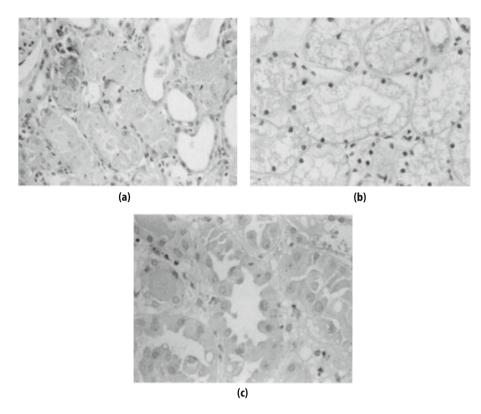


**Fig. 5.15 (a)** Congestion of the glomerular capillary loops. **(b)** Complete infarction in an area of cortical necrosis. **(c)** Fibrin thrombi within the loops. **(d)** Dilated Bowman's space with granular material and prominent epithelial cells.

Macroscopically, the kidneys are affected bilaterally and show an increase in size/weight. This, however, may be difficult to detect in the presence of pre-existing renal disease. Assuming that the kidneys are "normal", the capsules are under tension and the cut surfaces are oedematous (Fig. 5.13).

The cortex is often seen to be pale with dark congested medulla. Uni- or bilateral foci of cortical necrosis may be seen with the extreme end of the spectrum being complete cortical necrosis (Fig. 5.14).

On microscopic examination there are usually no changes seen to the glomeruli although in the very early stages, capillary congestion may be a feature, followed by a relatively bloodless appearance<sup>28</sup>. Fibrin thrombi may be seen within the capillary loops and in the case of cortical necrosis, glomerular infarction is seen. The Bowman's space may be dilated, which has been hypothesised to be due to reflux from the proximal tubules<sup>29</sup>. The cells lining the capsule may in turn be prominent and increased in number, having an appearance similar to those of the proximal tubules. Finally, the space frequently contains eosinophilic granular material (Fig. 5.15).



**Fig. 5.16** (a) Acute tubular necrosis. The lumen of the tubules is full of granular amorphous material with flattening of the epithelial cells. (b) Cloudy swelling. (c) During regeneration, mitotic figures are seen within the epithelial cells. ((a) and (c) courtesy of Professor P. Furness, University of Leicester, England).

A variety of changes have been reported to occur to the tubules in acute tubular necrosis depending upon the time course between the onset of symptoms and death. Many authors report that the changes associated with shock are mild compared to those of other causes of acute tubular necrosis, with epithelial cell necrosis been limited to single cells<sup>30</sup>. By day three following the insult the proximal tubules are frequently dilated with flattened epithelium although occasionally the epithelial cells may show marked swelling (Fig. 5.16). The normal Period Acid Schiff (PAS) positive border is lost and eosinophilic granular material is seen within the lumen. During the recovery phase mitotic figures are seen within the epithelial cells.

The distal tubules also show dilatation and flattening of the epithelial cells. In addition to this the lumen contain pigmented, granular or hyaline casts. Casts may also be seen within the urine on cytology (Fig. 5.18). The interstitium is expanded due to the presence of oedema and an inflammatory cell infiltrate may be seen.

Fibrin thrombi are found infrequently within the kidney in shock. When present, in the absence of cortical necrosis they are usually found within the glomerular arterioles but may also be seen occasionally in the venules and arcuate veins (Fig. 5.17). Even when they occur in large numbers they can undergo complete lysis within one week of the insult.

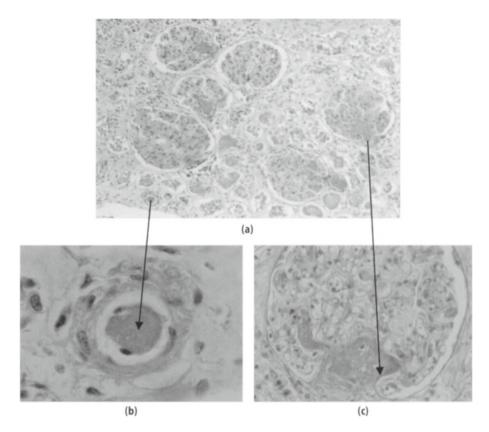


Fig. 5.17 (a) Extensive fibrin thrombi both within vessels (b) and within glomeruli (c).

As with the lungs, the differentiation between ante-mortem shock and post-mortem autolysis in the kidneys may be extremely difficult and at times almost impossible. The onset of the changes start within minutes of death and these changes may appear similar to those of true ante-mortem pathology. The time sequence can be summarised as follows<sup>4,31,32</sup>:

- In the glomeruli, the endothelial and mesangial cells show rapid autolysis.
- Within minutes of death the epithelium of the proximal tubules show signs of swelling with contraction of the lumina.
- Changes to the collecting tubules are delayed for up to 8 hours.
- Cellular debris is seen within the tubules at 24 hours.
- By 48 hours the proximal tubules are distended by amorphous eosinophilic material.

Thus, one has to consider the clinical history and the time between the insult and death. The weight of the kidneys and their macroscopic appearance must be taken into account as they do not swell or increase in weight purely due to post-mortem autolysis. However, these parameters may be of limited use if the kidneys are already shrunken due to previous natural disease. The microscopic appearances

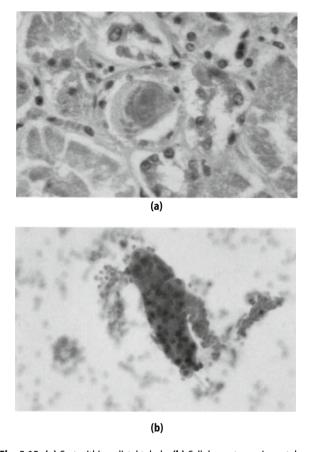


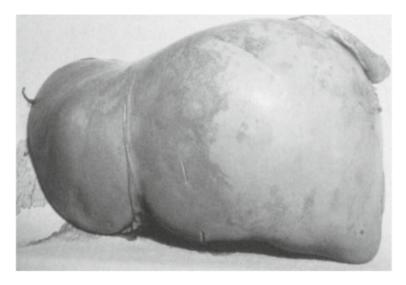
Fig. 5.18 (a) Cast within a distal tubule. (b) Cellular cast on urine cytology.

may be of limited use but one should consider the cellular appearances in relation to the ante- and post-mortem time periods and which components of the tubular systems are involved. The presence of infarction, fibrin thrombi, myoglobin or cellular regeneration will all assist in the differentiation between ante- and post-mortem change.

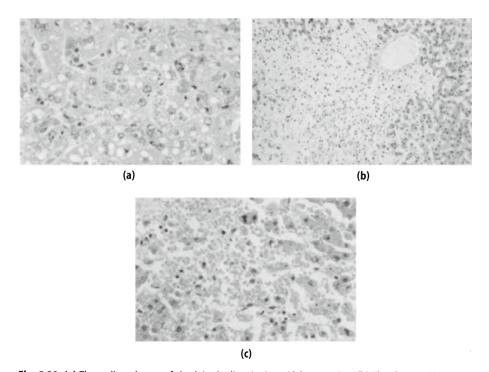
#### The Liver

The principal site affected in all types of shock in the liver is zone 3 of the acinus. Lesions have been reported to occur in 46.1% of cases of hypovolaemic shock, 56.3% of cardiogenic shock and 32% of septic shock although some authors believe that the liver is affected in all cases.

Clinically, the patient may become jaundiced, especially if there is pre-existing liver disease. Macroscopically, the liver is increased in weight and swollen. The capsule remains smooth and yellow discolouration due to fatty change may be prominent (Fig. 5.19).



**Fig. 5.19** The macroscopic appearance of the liver in shock. The capsule is tense and shows blotchy discolouration due to the presence of fat within the parenchyma.



**Fig. 5.20** (a) The earliest change of shock in the liver is sinusoidal congestion. (b) The characteristic asymmetric necrosis of shock liver. (c) Focal haemorrhage in areas of liver necrosis.

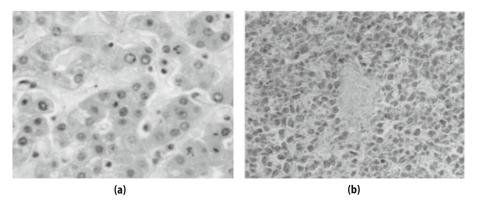
Ultrastructurally, necrosis may be seen as early as 2 hours after the onset of shock. However, microscopically the changes are seen within the first 24 hours and consist principally of hepatic necrosis. This is centred around the central vein and involves zone 3 but sometimes extends to involve zone 2. Acinar architecture and the periportal areas are unaffected. Experiments in dogs and rats during haemorrhagic shock show the first feature, as with other organs, to be that of sinusoidal congestion and engorgement (Fig. 5.20(a)). This is then followed by necrosis, which is often characterised by an asymmetrical pattern of involvement with no sinusoidal enlargement and no inflammation in the early stages<sup>33</sup> (Fig. 5.20(b)). This asymmetrical distribution can be used to differentiate the changes from congestive cardiac failure where the zone 3 involvement tends to be symmetrical<sup>34</sup>. In the liver, fibrin thrombi are rare, occurring in 0.5% of cases in one series. Large areas of haemorrhage are also uncommon although small areas of haemorrhage may be seen in association with areas of necrosis (Fig. 5.20(c)).

Septic shock may affect the excretion of conjugated bilirubin. This may lead to cholestasis with bile duct proliferation, cholangitis and bile concreations within small ducts<sup>35</sup>. Other non-specific changes may also be seen in all types of shock including fatty change and Kupffer cells hyperplasia<sup>36,37</sup>.

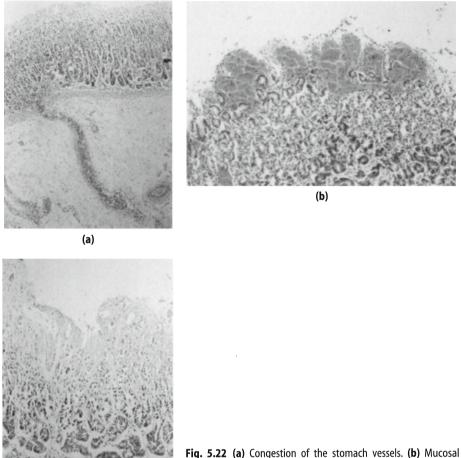
After 48 hours the liver will begin to repair. The affected zones will now see an infiltrate of neutrophils. These last for a few days before being replaced by macrophages which remove the necrotic liver cells to leave an empty reticulin framework. Liver regeneration will then occur with mitotic figures observed within regenerating hepatocytes (Fig. 5.21(a)).

The differentiation between the effects of shock and those of post-mortem autolysis of the liver are, as for the kidney, extremely difficult, if at times impossible. Detailed studies have been carried out in animals which show the following sequence of changes to occur<sup>38</sup>:

- By 2 hours swelling occurs within the mitochondria.
- At 4 hours the mitochondria become indistinct.
- At 6 hours breaks occur in the reticulin fibres and the nuclei distend.
- At 4 days the cell margins are no longer visible, reticulin fibres are no longer detectable and Kupffer cells are pyknotic.
- At 8 days only cell shadows are distinguishable.
- At 215 days only the elastic tissue remains.



**Fig. 5.21** (a) Mitotic figures within an area of regenerating liver parenchyma. (b) Post-mortem autolysis. Note the symmetrical appearance compared to that of shock illustrated in Fig. 5.20b.



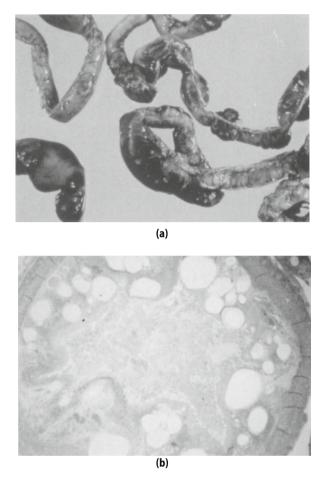
petechae. (c) Necrosis of the outer half of the mucosa without evidence of inflammation.

Despite this problem, the most useful change to aid distinction between the two processes is that the autolytic picture, as with cardiac failure, tends to show a symmetrical picture and thus asymmetrical areas of necrosis in the early post-mortem period may suggest the effect of shock rather than pure post-mortem autolysis (Fig. 5.21(b)).

#### **The Gastrointestinal Tract**

(c)

The stomach, small intestine and large intestine may all be affected by shock: septic in 26% of cases, cardiogenic in 16.2% and hypovolaemic in 8.8%. The principal

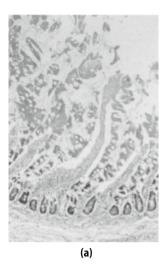


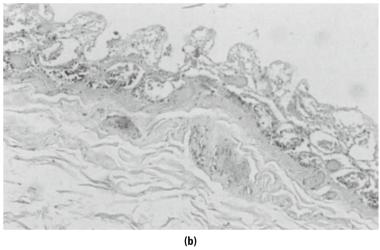
**Fig. 5.23 (a)** Necrotising enterocolitis **(b)** The submucosal air-filled cysts of pneumotosis intestinalis (Both images courtesy of Dr Variend, Sheffield Children's Hospital, England).

factors involved in the development of the lesions are hypotension, vasoconstriction of the splanchnic vessels and fibrin thrombi<sup>39</sup>.

The stomach may show changes 3–5 days after the triggering event although the onset may be delayed for up to 3 weeks. The fundus and body are the sites usually affected, showing a range of macroscopic changes from congestion to petechae to erosions to frank ulceration. Microscopically the lesions are confined to the outer half of the mucosa (except were true ulceration occurs) and comprise of oedema, vascular congestion, loss of surface epithelium and glands, haemorrhage and fibrin but again, as with the liver, the process lacks inflammation (Fig. 5.22).

The small and large intestines may also show a spectrum of changes which are termed ischaemic colitis<sup>40-43</sup>. These range from congestion and oedema with a macroscopic dusky red mucosal, friable wall and altered blood in the lumen to villous, mucosal or transmural necrosis/ulceration. Fibrin thrombi are frequent although an inflammatory infiltrate is not a feature until 2 days after the onset of the lesions.

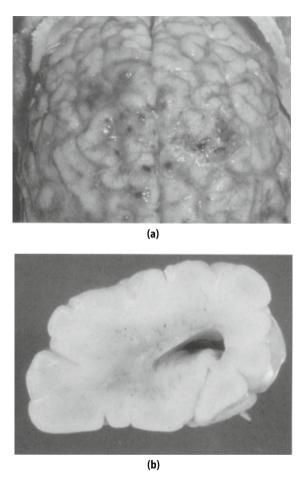




**Fig. 5.24** (a) Post-mortem small intestine showing: (b) large-intestine mucosa in a case of septic shock illustrating the difficulty in differentiating shock from post-mortem autolysis. Vascular congestion is present although there are no other features to assist one in this case.

In infants the extreme change of necrotising enterocolitis may occur<sup>44,45</sup> (Fig. 5.23(a)). This change may be associated with gas cysts (pneumotosis intestinalis) in the submucosa and subserosal areas which in turn may lead to perforation and peritonitis<sup>46,47</sup> (Fig. 5.23(b)). Healing lesions may lead to extensive submucosal fibrosis and the formations of strictures.

The differentiation between post-mortem changes of the small-intestinal villi and shock again will cause problems as villous oedema and desquamation of epithelial cells can occur within 4–5 hours of the onset of shock. In the post-mortem period there is rapid loss of the small and large intestinal mucosal into the lumen of the bowel (Fig. 5.23). The lack of vascular engorgement and fibrin thrombi can be



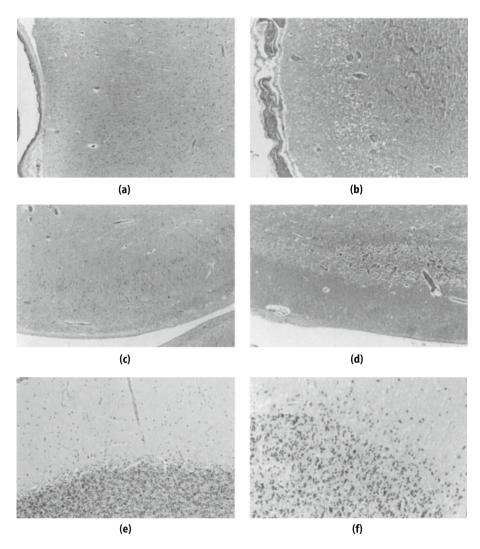
**Fig. 5.25 (a)** Haemorrhages to the superior external surface of the cerebral cortices in the presence of DIC. **(b)** Periventricular leukomalacia in a neonate (Courtesy of Dr Variend, Sheffield Children's Hospital, England).

utilized to try and differentiate between this and the effect of shock although the differentiation between the two processes may not be possible.

#### The Brain

The brain is not a common site for lesions in shock. The commonest change that does occur is that of hypoxic brain injury. In one series pathology was found in 5.9% of hypovolaemic shock, 4.1% of cardiogenic shock and 3% of septic shock cases. The reason behind the relative lack of cases is thought to be the brain's autoregulatory ability to maintain a cerebral blood flow. Changes are thus seen when the systolic pressure falls below 65 mmHg.

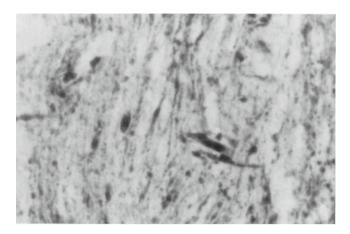
The macroscopic appearance of the adult brain may show a spectrum of changes from no macroscopic lesions to areas of infarction, commonly in the border zones



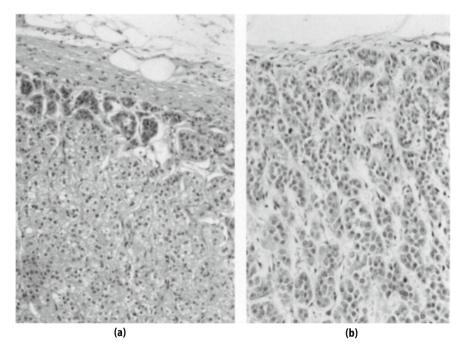
**Fig. 5.26** (a) Normal cortex. (b) Hypoxic cortex. (c) Normal hippocampus. (d) Hypoxic hippocampus (e) Normal cerebellum. (f) Hypoxic cerebellum. (Courtesy of Professor W. Timperley, formerly of the Department of Neuropathology, Royal Hallamshire Hospital, Sheffield, England).

between the cerebral arteries ("watershed infarcts"). If disseminated intravascular coagulation is present then haemorrhages may be seen to the external and cut surface of the brain (Fig. 5.25 (a)). In the neonate, periventricular leukomalacia may be observed<sup>48</sup> (Fig. 5.25 (b)).

On microscopy the brain shows the typical changes of hypoxic injury. The neurons are damaged in preference to the astrocytes and oligodendrocytes with the areas involved been the third cortical area particularly of the parietal and occipital lobes, Sommer's sector and the end folium of the hippocampus, the outer halves of the caudate nucleus and putamen, the anterior nuclear complex of the thalamus,



**Fig. 5.27** Axonal bulbs in a case of haemorrhagic shock. There was no clinical history of head trauma. Immunohistochemical staining against  $\beta$  amyloid precursor protein.



**Fig. 5.28 (a)** Normal adrenal cortex showing fat within the cells of the zona fasciculate. **(b)** Fat depletion of the adrenal gland in shock.

the Purkinje and basket cells of the cerebellum and in extreme cases the reticular zones of the substantia nigra and inferior olives of the brain stem (Fig. 5.26). In those that survive at least 2 hours after the initial insult, immunohistochemical staining against  $\beta$  amyloid precursor protein can show both neuronal body and axonal staining with axonal retraction bulbs occurring in the same distribution typically described for that of diffuse axonal injury<sup>49,50</sup> (Fig. 5.27).

The second change that may be seen in these cases is that of fibrin thrombi within the blood vessels, which in turn show surrounding ring haemorrhages. The vessels will undergo fibrinoid change and show an infiltrate of neutrophil polymorphs. In the first 6–8 hours post-mortem the brain is relatively resistant to autolytic change<sup>4</sup>. After this time autolysis will set in, ultimately resulting in difficultly in removing the brain and examination due to liquifactive change. All pathologists undertaking autopsy examinations will have experienced the autolytic brain which quite literally pours out of the cranial cavity. By the time that it reaches this point, further examination is often futile. Ring haemorrhages and fibrin thrombi are again not a feature of early post-mortem autolysis. The pattern of hypoxic change as described above may also be used to differentiate between ante-mortem hypoxia and post-mortem autolysis.

#### The Adrenal Glands

Early authors interpreted the changes observed in the adrenal gland in shock in terms of the disorders with which the change was associated, rather than considering the observation as part of a more systemic reaction to shock. The best example of this is the haemorrhage seen within the adrenal gland in association with meningo-coccus septicaemia. This was considered to be peculiar to infection with meningo-coccal infection and was described as Waterhouse-Friderichsen syndrome<sup>51,52</sup>.

However, as has been detailed above, haemorrhage is in fact one of four manifestations of shock of all causes seen within the adrenal gland: the other three changes being lipid depletion, fibrin thrombi and necrosis. Each change may occur on its own or in combination with another manifestation. In the adrenal gland the lesions are usually bilateral, occurring in up to 14.1% of all cases of shock. The plasma corticosteroid and adrenalin levels are usually elevated, suggesting that adrenal injury may, in the majority of cases, be focal rather than diffuse although both patterns may be seen.

In the adult, the most frequently observed change is that of lipid depletion of the zona fasciculate of the cortex. Although not specific to shock, as lipid depletion may occur as a consequence of any cause of stress, it can be seen as focal or complete involvement of the zona. Instead of the normal lipid-rich cells one sees compact aggregates of lipid-depleted cells (Fig. 5.28). Cytolytic degenerative changes may also be seen within the same zona with resulting spaces between the residual cellular columns.

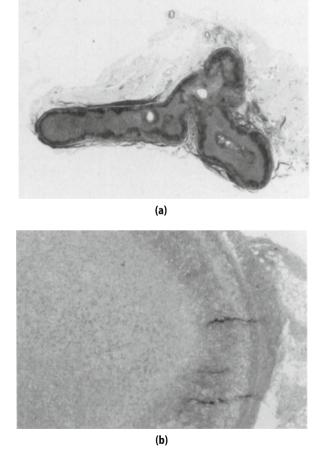
In the stillborn, lipid depletion is seen to the foetal cortex with sparing of the definitive cortex. The sparing of the inner foetal cortex leads to the so-called "clear cell reversal" pattern. Focal degeneration of the cortical cells as a result of prenatal stress produces a pseudofollicular change to the definitive cortex.

The degree of haemorrhage can vary from focal cortical congestion with extravasation of red cells to whole organ involvement. It is usually bilateral with unilateral involvement being rare. The haemorrhage may be confined to the cortex although in severe cases it can extend beyond the capsule into the surrounding adipose tissue (Fig. 5.29).

As with haemorrhage, the degree of cortical necrosis may vary between focal collections of necrotic cells to whole cortical infarction (Fig. 5.30). Necrosis is usually associated with the more severe end of the spectrum of haemorrhage although it can be seen in the absence of haemorrhage. Under this circumstance 50% of cases are associated with fibrin thrombi.

Fibrin thrombi may be seen with or without haemorrhage or necrosis. They are usually seen within the adrenal sinusoids although they may also occur to the central vein, especially when there is massive haemorrhage when thrombi may be found in up to 75% of cases. Smaller degrees of haemorrhage are associated with thrombi in two-thirds of cases.

In the post-mortem period the adrenal gland will undergo autolysis in accordance with other organs. Although the glands will retain their macroscopic architecture, transverse cuts across the gland will show loss of cohesion of the medulla. The



**Fig. 5.29** (a) Diffuse cortical haemorrhage of the adrenal gland. (b) A band of cortical haemorrhage with extension into the peri-adrenal fat.

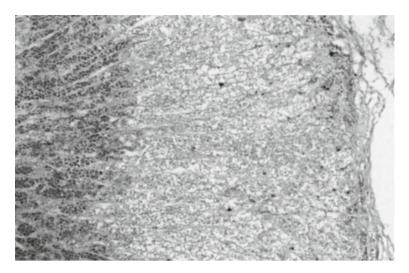


Fig. 5.30 Extensive cortical necrosis of the adrenal gland in shock.

characteristic yellow colour of the cortex is maintained in the early post-mortem period. To date, no studies are reported as to how long the morphology of the adrenal gland remains after death, although from experience good cellular definition can persist for several days. Haemorrhage and fibrin thrombi are not a feature of post-mortem autolysis of the adrenal gland and again their presence should draw one's consideration to a systemic ante-mortem cause for these changes.

## **The Pancreas**

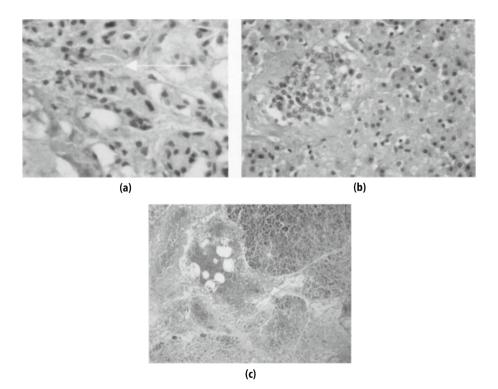
The principle pathology of the pancreas and shock is that of acute pancreatitis. As it is indistinguishable from other forms of acute pancreatitis the changes may be due to shock or may be the cause of shock. As with other organs of the body the features are those of haemorrhage, necrosis and fibrin thrombi<sup>53-60</sup>.

Pancreatitis as a complication of shock is uncommon, with an occurrence of 6.9% in hypovolaemic shock, 6% in septic shock and 2.5% in cardiogenic shock reported. When present, the lesions may be associated with the presence of acute tubular necrosis (ATN): 50% of cases present in the presence of ATN in hypovolaemic shock, 9% in its absence.

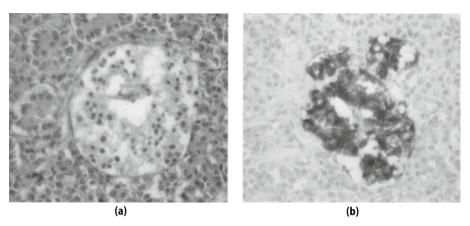
The exocrine acinar is the site of the pathological changes with cell damage to the Islets of Langerhans being rare (0.04% on their own), having only been reported to date to occur in newborns and young children<sup>61-63</sup>. Following an episode of hypovolaemia, changes may be seen approximately 1 hour from the onset of the event. The first microscopic change seen is that of interstitial and intralobular oedema although electron microscopic studies have shown ultra-structural cellular damage to proceed microscopic changes. This is followed by the onset of haemorrhage. At this stage the gland is swollen and there is capillary stasis. This may then lead to fulminent necrosis and the typical appearance of acute pancreatitis. The Islet cells are uninvolved and can be distinguished from the surrounding necrotic exocrine pancreas. Microthrombi may be found in the vicinity of areas of necrosis (Fig. 5.31).

As stated above, involvement of the Islets of Langerhans is rare with all case reports occurring in children. To date, these observations have not been reported in adults although the illustrations shown in Fig. 5.32 are from adult cases. The Islet cells are reported to show coagulative necrosis without an inflammatory reaction. A central core of necrotic cells may be seen to be surrounded by a peripheral rim of intact cells. Other changes reported to the endocrine pancreas include hydropic change in the form of cloudy swelling, vacuolar degeneration and haemorrhage. Immunohistochemical staining shows intense staining of the necrotic cells for insulin and glucagon.

In the post-mortem period the pancreas will undergo rapid autolytic changes. In fact, it is often the first organ in which such changes can be observed. Mukai reported the sequence of post-mortem changes in 181 humans and 57 rabbits in relation to the time since death, and graded the changes into six phases, although to date, due to the large degree of variability of the onset of these changes, most pathologists do not rely on this scheme for assessing the timing of any changes within the pancreas<sup>64</sup>. Unlike the changes of shock, both the exocrine and endocrine components of the gland are affected by the autolytic process (Fig. 5.33). Although the onset is often focal, ultimately the entire gland is affected such that just the ghost outline of the cells can be seen. Unlike that of fulminant shock, no inflammatory component is present although a degree of focal haemorrhage can be seen.



**Fig. 5.31 (a)** Fibrin thrombi (arrow) within the pancreas. **(b)** Necrosis with sparing of the islets. **(c)** Haemorrhage of the pancreatic parenchyma and surrounding fat.



**Fig. 5.32** (a) Islet of Langerhans showing necrosis in a case of haemodynamic shock in an adult. (b) Intense staining against insulin with immunohistochemical staining.

# **The Pituitary Gland**

In the latter part of the twentieth century pituitary glands were often removed without examination for the purpose of growth hormone extraction. However, in more modern times they are often left in situ within the sella turcica. Thus, it is difficult to estimate the percentage of pituitary glands affected by shock although Plaut

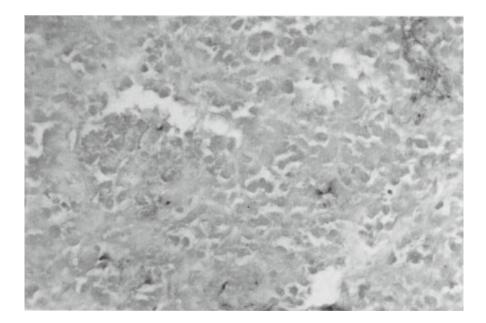


Fig. 5.33 Post-mortem pancreas showing involvement of both the endocrine and exocrine parts of the gland.

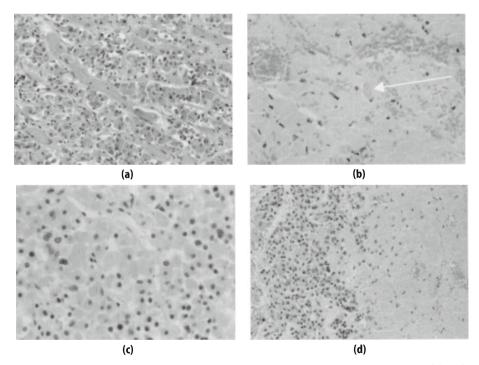
and Kovacs have estimated that in 1–8% of unselected autopsies, foci of necrosis may be found within the anterior lobe. Saeger and Hanker in two separate series estimated 8% and up to 20% of anterior lobes to show micro-necrosis<sup>65–73</sup>.

Both the anterior and posterior lobes can be affected by shock although the latter is extremely uncommon. The best example of the effects of shock on the pituitary gland were described by Sheehan in relation to post-partum haemorrhagic shock. As with the pancreas, the pathology seen in the pituitary in shock is not specific for shock and can be seen following head injury, raised intra-cranial pressure or in those on ventilatory support. The extreme variant of haemorrhage into the gland, that of pituitary apoplexy, is rare in shock, being more often associated with pituitary adenomas.

In the case of the pituitary, the principal pathology is that of haemorrhage with or without necrosis and fibrin thrombi of the adenohypophyseal sinusoids. The time sequence of changes as described by Sheehan is as follows:

- 6–12 hours Vascular dilatation and congestion with blood cells.
- 12-15 hours Separation of basement membrane.
- 15 hours Necrosis.
- 25 hours Red cell disruption. Vessels appear empty.

This sequence of events is illustrated in Fig. 5.34. Whilst these changes affect the anterior lobe there is always a band of unaffected, viable subcapsular cells. Ninety percent of the lobe can be affected before the onset of clinical symptoms.



**Fig. 5.34** (a) Vascular congestion. (b) Fibrin thrombi (arrow). (c) Single cell necrosis. (d) Sparing of the subcapsular area with central necrosis.

The posterior lobe is rarely affected by shock with the supraoptico- and parventriculo-hypophyseal tracts occasionally affected. Magnocellular nuclear atrophy is also reported to occur to the posterior lobe.

Post-mortem changes of the pituitary gland start to occur to the anterior lobe within 30 minutes of death. Macroscopically the pituitary becomes diffluent within the sella turcica, making removal difficult without resulting damage to the gland. Autolysis starts in the gamma cells with the alpha cells undergoing autolysis by 36 hours. Within the posterior lobe there may be petechial haemorrhage, which is considered to be a common post-mortem artefact<sup>74</sup>.

## The Lymphoreticular System

The spleen and lymph nodes are not usually considered to be organs that show changes in cases of shock, which accounts for the lack of publications touching upon this system. There are no reported publications concerning the pathology of the lymphoreticular system in shock in adults but occasional publications concerning the changes to the germinal centres of the spleen and lymph nodes do exist for children relate predominately to septic shock. Fifty percent of children aged 1 to 3 years in a series of 200 (80% died of "shock") reported by Howat and Variend showed changes to the follicles. It is, however, not possible to state how often changes are seen to this system and if they occur in other causes of shock as the data does not exist to date<sup>77</sup>.

The reported pathology of both the spleen and lymph nodes are nuclear fragmentation and epitheloid change of the follicles. This manifests as karyorrhexis and atrophy of the germinal centres (Fig. 5.35). It is thought that as the germinal centres are vascularized by terminal branches of afferent arterioles, they are particularly vulnerable to the effects of shock. These changes can be seen to other sites of lymphoid follicles including the germinal centres of the appendix.

In the post-mortem period, macroscopically the spleen will be become diffluent. In a study by Tappero it was shown that the autolytic changes started in the red



**Fig. 5.35** A follicle within the spleen from the series reported by Howat and Variend. (Courtesy of Dr Variend, Sheffield Children's Hospital, England).

pulp with good follicle stability for up to 30 days post-mortem<sup>78</sup>. By 60 days, nuclear staining of the follicles had deteriorated and by 120 days post-mortem, the nuclear staining of the follicles had almost gone. Nuclear fragmentation and epitheloid change is thus not a change attributable to autolysis and, if present, could be used to indicate a diagnosis of shock.

## **Other Organs**

Haemorrhage and fibrin thrombi may occur in many of the other organs of the body. Thrombi may be seen in the skin, manifesting externally as a purpuric rash. Haemorrhage is also reported to occur in the bladder in shock. These changes can be differentiated from post-mortem autolysis again as fibrin thrombi and haemorrhage are not a feature of autolysis even at these sites. Finally, spermatogonial necrosis of the testis has been reported to occur in up to 26% of infant males, with shock associated with congenital heart disease<sup>79</sup>.

## **Summary**

The principal features of shock to all organs of the body are those of vascular congestion and engorgement followed by necrosis, haemorrhage and the presence of fibrin thrombi. The pattern and distribution of these changes can be used to differentiated these changes from pure post-mortem autolysis although, depending upon the organ involved, how soon after the onset of shock the patient died and the post-mortem period, this task can be extremely difficult, if not at times impossible. Under these circumstances it is often the clinical history that one becomes more reliant upon rather than the post-mortem findings, which may be affected or masked by post-mortem changes.

#### References

- 1. Thompson D, editor. The concise Oxford dictionary. 9th ed. Oxford: Claredon Press, 1995.
- 2. May C. Lord Moran's memoir: Shell-shock and the pathology of fear. J R Soc Med 1998;91:95-100.
- 3. Reide U, Sandritter W, Mittermayer C. Circulatory shock: a review. Pathology 1981;13:299-311.
- Post-mortem Changes. In: Janssen W, editor. Forensic histopathology. Berlin: Springer-Verlag, 1984;13–46.
- 5. Pietra G. The lung in shock. Hum Pathol 1974;5:121-2.
- Katzenstein AA, Bloor CM, Leibow AA. Diffuse alveolar damage the role of oxygen, shock and related factors. Am J Pathol 1976;85:210–28.
- 7. Addis BJ. Diffuse alveolar damage. In: Corrin B, editor. Systemic pathology. vol. 10. The respiratory system. London: Churchill Livingston, 1995;55–68.
- 8. Corrin B. Lung pathology in septic shock. J Clin Pathol 1980;33:891-4.
- 9. Schlag G, Redl H. Morphology of the microvascular system in shock: lung, liver and skeletal muscles. Crit Care Med1985;13:1045–9.
- Orell SR. Lung pathology in respiratory distress following shock in the adult. APMIS Sect A 1971;79:65-76.
- 11. Moon VH. The pathology of secondary shock. Am J Pathol 1948;24:235-73.
- 12. Aabo K, Hansen KB. Megakaryocytes in pulmonary blood vessels. 1. Incidence at autopsy, clinicopathological relations especially to disseminated intravascular coagulation. APMIS. Sect A. 1978;86:285–91.
- 13. Pumphrey RSH, Roberts ISD. Investigating possible anaphylactic deaths. In: Burton J, Rutty G, editors. The Hospital Autopsy, 2nd ed. London: Arnold, 2001;147–58.

- 14. McGovern VJ. Shock. Pathol Ann. 1971;6:279-98.
- 15. McGovern VI. Shock revisited. Pathol Ann 1984;19:15-36
- 16. McGovern VJ, Tiller DJ. Shock. A clinicopathological correlation. New York: Masson, 1980.
- 17. Krausz T, Cohen J. Shock. In: McGee J O<sup>3</sup>D, Isaacson PG, Wright NA, editors. Oxford textbook of pathology. Oxford: Oxford University Press, 1992;550-51.
- 18. Shock. In: Janssen W, editor. Forensic histopathology. Berlin: Springer-Verlag, 1984;156-78
- 19. Evans TJ, Krausz T. Pathogenesis and pathology of shock. In: Anthony PP, MacSween RNM, editors. Recent advances in histopathology 16. London: Churchill Livingston, 1994;21–48.
- 20. Wiggers CJ. Physiology of shock. New York: Commonwealth Fund. 1950.
- 21. Hackle DB, Goodale WT. Effects of haemorrhagic shock on the heart and circulation of intact dogs. Circulation 1955;11:628–34.
- 22. Martin AM, Hackle DB. The myocardium of the dog in haemorrhagic shock. A histochemical study. Lab Invest 1963;12:77-91.
- 23. Lisagor P, Cohen D, McDonnell B, Lawlor D, Moore C. Irreversible shock revisited: mechanical support of the cardiovascular system: a case report and review. J Trauma 1997;42:1182-6.
- 24. McGovern VJ. Hypovolaemic shock with particular reference to the myocardial and pulmonary lesions. Pathology 1980;12:63-73.
- 25. Page DL, Caulfield JB, Kastor JA, DeSanctis RW, Sanders CA. Myocardial changes associated with cardiogenic shock. New Eng J Med 1971;285:133-7.
- 26. Todd GL, Baroldi G, Pieper GM, et al. Experimental catecholamine-induced myocardial necrosis. I. Morphology, quantification and regional distribution of acute contraction bands lesions. J Mol Cell Cardiol 1985a;17:317–38.
- 27. Davies MJ, Mann JM, editors. Systemic pathology, vol. 10. The cardiovascular system. Part B: Acquired diseases of the heart. London: Churchill Livingston, 1995;44–6
- 28. Dalgaad OZ. An electron microscopic study of glomeruli in renal biopsies taken from human shock kidney. Lab Invest 1960;9:364-6.
- 29. Waugh D, Schlieter W, James AW. Intraglomerular epithelial reflux: an early lesion of acute renal failure. Arch Pathol 1964;77:93.
- 30. Jennette JC, Olson JL, Schwartz MM, Silva FB, Heptinstall RH, editors. Acute renal failure. In: Heptinstall's pathology of the kidney, 5th ed. London: Lippincott William and Wilkins,1998.
- 31. Martines G. Autolisi postmortale del tubulo nefronico sperimentalmente provocata. Riv Patol Clin Sper 1964;5:263.
- 32. Osvaldo l, Jakson JD, Cook ML, Latta H. Reactions of kidney cells during autolysis. Light microscopic observations. Lab Invest 1965;14:603.
- 33. Birgens HS, Henriksen J, Matzen P, Poulsen H. The shock liver. Acta Med Scand 1978;204:417-21.
- 34. Lefkowitch JH, Mendez L. Morphologic features of hepatic injury in cardiac disease and shock. J Hepatol 1986;2:313–27.
- 35. Ishak KG, Rogers WA. Cryptogenic acute cholangitis assiociation with toxic shock syndrome. Am J Clin Pathol 1981;76:619–26.
- 36. Banks JG, Foulis AK, Ledingham IM, MacSween RNM. Liver function in septic shock. J Clin Pathol 1982;35:1249-52.
- 37. Cowley RA, Hawkins JR, Jones RT, Trump BF. Pathology and pathophysiology of the liver. In: Cowley RA, Trump BF, editors. Pathophysiology of shock, anoxia and ischaemia. Baltimore: Williams and Wilkins, 1982;285–300.
- 38. Muller PH, Willot M, Oliveira de SAFM, Debarge A. Aspects histologiques de l'autolyse hepatique. Acta Med Leg Soc (Leige) 1965;18:243.
- 39. Itoh M, Paulsen G, Guth PH. Hemorrhagic shock and acid gastric injury in the rat. Gastroenterology 1986;90:1103-10.
- 40. Bailey RW, Hamilton SR, Morris JB, Bulkley GB, Smith GW. Pathogenesis of non-occlusive ischaemic colitis. Ann Surg 1986;203:590-99.
- 41. Marston A, Pheils MT, Thomas ML, Morson BC. Ischaemic colitis. Gut 1966;7:1.
- 42. Bailey RW, Hamilton SR, Morris JB, Bulkley GB, Smith GW. Pathogenesis of non-occlusive ischaemic colitis. Ann Surg 1986;203:590-99.
- 43. McGovern VJ, Goulston SJM. Ischaemic enterocolitis. Gut 1965;6:213-20.
- 44 Kliegman RM, Fanaroff AA. Necrotising enterocolitis. New Eng J Med 1984;310:1093-103.
- 45. Whithead R, editor. Necrotising enterocolitis of the newborn. In: Gastrointestinal and oesophageal pathology. London: Churchill Livingstone, 1995;699–701.
- 46. Gilbert-Barness E, editor. Neonatal necrotising enterocolitis. In: Potter's pathology of the fetus and infant, vol 2. London: Mosby, 1997;803–05.
- 47. Wigglesworth JS, editor. Intestinal infarction and necrotising enterocolitis. In: Perinatal pathology. London: WB Saunders, 1984;266–70.

- 48. Auer RN, Sutherland GR. Hypoxia and related conditions. In: Graham DI, Lantos PL editors, Greenfield's neuropathology, 7th ed. London: Arnold, 2002;233–80.
- 49. Kaur B, Rutty G, Timperley W. The possible role of hypoxia in the formation of axonal bulbs. J Clin Pathol 1999;52;203–09.
- 50. Harrington D, Rutty GN, Timperley WR. β amyloid precursor protein axonal bulbs may form in non-head-injured patients. J Clin Forensic Med 2000;7:19–25.
- 51. Councilman WT, Mallory FB, Pearce RM. A study of the bacteriology and pathology of 220 fatal cases of diptheria. J Boston Soc Med Sci 1900;5:139–319.
- 52. Greendyke RM. Adrenal haemorrhage. Amer J Clin Pathol 1965;43:210-15.
- 53. Warshaw AL, O'Hara PJ. Susceptibility of the pancreas to ischaemic injury in shock. Ann Surg 1978;188:197–201.
- 54. Pfeffer RB, Lazzarini-Robertson A, Safadi D, Mixter G, Secoy CF, Hinton JW. Gradations of pancreatitis, edematous, through hemorrhagic, experimentally produced by controlled injection of microspheres into blood vessels in dogs. Surgery 1962;51:764–9.
- 55. Feiner H. Pancreatitis after cardiac surgery. Am J Surg 1976;131:684-8.
- 56. Seemayer TA, Osbourne C, De Chadarevian J-P. Shock-related injury of pancreatic Islets of Langerhans in newborn and young infants. Hum Pathol 1985;16:1231-4.
- 57. Bernstein J. Renal tubular and pancreatic islet necrosis in newly born infants. Am J Dis Child 1958;96:705–10.
- 58. Gmaz-Nikulin E, Plamenac P, Hegewald G, Gaon D. Pancreatic lesions in shock and their significance. J Pathol 1981;135:223-36.
- 59. Barzilai A, Ryback BJ, Medina JA, Toth L, Dreiling DA. The morphological changes of the pancreas in hypovolemic shock and the effect of pretreatment with steroids. Int J Pancreatol 1987;2:23-32.
- Jones RT, Garcia JH, Mergner WJ, Pendergrass RE, Valigorsky JM, Trump BF. Effects of shock on the pancreatic acinar cell. Cellular and subcellular effects in humans. Arch Pathol 1975;99:634–44.
- 61. Emery JL, Bury HPR. Involutionary changes in the Islets of Langerhans in the foetus and newborn. Biol Neonat 1964;6:16–25.
- 62. Jaffe R. Pancreatitic Islets in Shock. In: Stocker JT, Dehner LP, editors. Pediatric pathology, 2nd ed, vol 2. London: Lippincott William and Wilkins, 2001;824.
- 63. Betz EH. Les modifications des glandes endocrines au cours des etats de shock. Acta Anaesthesiol Belg 1968;3:224–33.
- 64. Mukai S. Determination of post-mortem interval by histological examination of pancreas. Jap Z Leg Med 1955;9:19.
- 65. Kovacs K. Necrosis of anterior pituitary in humans. Part 1. Neuroendocrinology 1969;4:170-99.
- 66. Kovacs K. Necrosis of anterior pituitary in humans. Part 2. Neuroendocrinology 1969;4:200-41.
- 67. Kovacs K. Adenohypophysial necrosis in routine autopsies. Endocrinologie 1972;60:309-16.
- 68. Plaut A. pituitary necrosis in routine autopsies. Am J Pathol 1952;28:883-99.
- 69. Saeger W, Hanke D. Nekrosen der Hypophyse und ihre Beziehung zum Schock. Verh Dtsch Ges Pathol 1978;62:301.
- 70. Sheehan HL, Murdoch R. Post-partum necrosis of the anterior pituitary; pathological and clinical aspects. J Obst Gynaec Brit Emp 1938;45:456–90.
- 71. Sheehan HL The repair of post-partum necrosis of the anterior lobe of the pituitary gland. Acta Endocrinol 1965;48:40-60.
- 72. Sheehan HL, Davis JC. Pituitary necrosis. Br Med Bull 1968;24:59-70.
- 73. Horvath E, Scheithaver BW, Kovacs K, Lloyd RV. Hypothalamus and pituitary. In: Graham D, Lantos P, editors. Greenfields neuropathology. 7th ed, vol. 1. London: Arnold, 2002;1024.
- Latka H, AdamW. Hyaluronidase als Ursache fruher postmortaler Verandererunggen der Hypophyse. Dtsch Z Ges Gerichtl Med 1953;42:32.
- 75. Kojima M, Takahashi K. Pathological study on the changes of lymphatic tissues in shock with special reference to the secondary nodule lesion. Acta Pathol Jpn 1971;21:387–403.
- Kojima M, Takahashi K, Yamaguchi A, Sue A, Terashima K. Experimental analysis on pathogenesis of the secondary nodule lesion in lymph nodes induced in shock. Acta Pathol Jpn. 1971;21:405–14.
- 77. Howat AJ, Variend S. Nuclear fragmentation and epitheliod change of germinal centres in the lymphoid tissue of child deaths. Pediatr Dev Pathol 1986;5:125–34.
- 78. Tappero P. Contributo allo studio tanatologico della milza. Minerva Med Leg (Torino) 1969;89:12.
- 79. Coen R, McAdams AJ Visceral manifestations of shock in congenital heart disease. Am J Ds Child 1970;199:383–9.

# 6. Radiological Investigations in Autopsy Practice

C.A. Wilson, A.K. Bonner, G.N. Rutty

#### Introduction

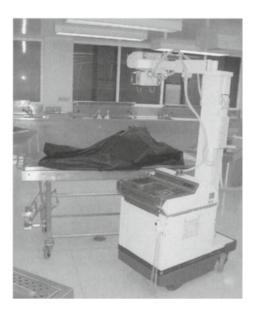
The aim of this chapter is to give an overview of the application of imaging techniques to autopsy practice. The intention is to discuss the relative merits of the various imaging techniques which may be used as an adjunct to post-mortem examinations. It is also intended to provide useful hints and practical advice to radiographers, radiologists, pathologists and mortuary staff engaged in imaging the deceased. The indications for post-mortem imaging are divided into those where it is considered mandatory, namely in cases involving the investigation of paediatric deaths, gunshot wounds and deaths from explosions, and those where imaging may be helpful, for instance decomposed, burned or severely traumatised bodies<sup>1-3</sup>.

#### **Techniques Available for Post-mortem Imaging**

In clinical practice, imaging options include plain x-rays, computerised tomography, angiography, magnetic resonance imaging and ultrasound. Each has its advantages and disadvantages in a clinical setting, but the nature of post-mortem imaging places particular constraints on the choice of modality. Not least of these is the availability of equipment, which is often complicated by the geography of the mortuary and the availability of radiographers who are familiar with post-mortem imaging.

#### **Plain X-rays**

Plain x-rays are still the most commonly used modality. There are a number of reasons for this, which include the ready availability of portable machines which can be taken into all but the most archaic of mortuaries. Larger mortuaries may either have a resident machine, although these are often of older design, or direct access to a clinical radiology department which may be in close proximity to the mortuary. Though older equipment may be obsolete for clinical purposes, if x-rays are only required for foreign body location then the image quality may be more



**Fig. 6.1** Mobile x-ray machine in use in a mortuary.

than adequate. Plain x-ray films provide a permanent record, and although this may give rise to some archiving problems, they are familiar to pathologists, radiologists and radiographers alike.

The most common types of equipment available for mortuary use are medium to high frequency mobile x-ray machines, capable of producing a full range of exposures needed for post-mortem imaging. The basic requirements are for a machine which can provide a wide range of exposures from soft tissue views to lateral chest or abdominal views (Fig 6.1).

#### **Computerised Radiography**

Most hospitals are now taking advantage of advances in computerised radiography (CR) and moving towards a filmless department. CR has a number of significant advantages when compared with conventional plain films. The image quality of CR is undoubtedly superior to that of plain films. Soft tissue information can be so detailed that a projectile path may be traced, something which is far harder to achieve with conventional media. CR software technology allows manipulation of digital information, allowing fine-tuning to produce an optimum image. Areas can be specifically enhanced or magnified, and if desired the original and manipulated images can be displayed side by side for comparison.

The majority of post-mortem images are taken to provide information for the pathologist, and therefore are seldom used as evidence in court. However, in some circumstances, for instance in fractures found in a paediatric skeletal survey or adult head injuries, post-mortem imaging may provide crucial evidence in legal proceedings. One possible drawback of CR is that the potential for digital image manipulation may be used to question the authenticity of images used in

court. In practice, every alteration to the original image is recorded on the computer hard drive, so advances in computer security and forensic computing have made it possible to refute any allegations of image manipulation.

Another advantage of CR is that archiving is simplified, and the images can be easily networked over secure sites to any designated person. This means that providing there is network access the pathologist can consult the radiographer or seek a second opinion from the mortuary, with minimal disruption to the work patterns of either party. Because digital images are more easily accessible than plain x-rays, their presentation can be enhanced by multimedia techniques such as the incorporation of text or graphics, which are useful for teaching purposes.

The major limitation of CR is "white out" on certain projections, where adjacent areas of high and low density are difficult for the digital readers to process. One example of this is the lateral chest view, where overlying limbs and non-uniform inflation of lung tissue can cause problems for the CR system. These problems can usually be overcome by careful radiographic technique, good positioning, collimation and post-processing factors.

#### **Fluoroscopy**

Fluoroscopy has the advantage of providing a real-time image to guide the pathologist searching for radio-opaque foreign bodies, which is useful in situations where there are multiple foreign bodies. The types of cases in which fluoroscopy might be considered are deaths from military ordnance, terrorist explosions or in multiple gunshot wounds, especially if missiles have fragmented. For this reason, fluoroscopy was used to great effect in the investigation of bodies recovered from mass graves in the former Republic of Yugoslavia. It has also been used with great success to examine the ground fill from a clandestine grave in the UK for projectiles, saving the investigating team many days of sieving of bags of heavy clay (Fig 6.2).

The drawbacks of this technique are primarily the limited availability of fluoroscopy machines in mortuaries, and the narrow field of view available. Also, not all machines, particularly the older equipment likely to be available for postmortem use, have a hardcopy facility. The problem of the narrow field of view and the lack of hardcopy can both be overcome by obtaining standard views with plain x-rays prior to fluoroscopy. With regard to the availability of equipment, since the main role of post-mortem fluoroscopy is simply foreign-body location, high resolution is not of prime concern. Therefore, older machines which are no longer suitable for clinical use may be more than adequate for mortuary use, and radiology departments should be encouraged to consider a new home for their outdated equipment in the mortuary.

#### Computerised Axial Tomography (CT) Scanning

The bulk of a CT scanner means that this is a piece of equipment where the body really does have to go to the equipment rather than vice versa. It is understandable that clinicians are not keen to have dead bodies passing through their scanners, which, coupled with the high clinical demand for the scanners, means that while an effective technique, CT scanning is unlikely to be a modality of choice for practical reasons. Also, the shadowing caused by metallic foreign bodies can cause



Fig. 6.2 An image intensifier in use in a mortuary examining bags of grave fill for projectiles.

extreme artefact in gunshot cases, although some of the artefact can be modified digitally and the image can be used to trace the path of the projectile through the body. If CT scanning is available, the entire body can be scanned extremely rapidly (a "trauma" scan), prior to magnified serial sections of areas of interest. Other advantages of CT scanning include the fact that the body does not have to be removed from the sealed body bag prior to imaging, and that the images can be produced as both hardcopy or on compact disc (CD) (Fig 6.3).

If available, ante mortem CT images of head injuries should always be reviewed prior to the autopsy as they will inform the pathologist of the location and nature of any intracranial pathology. This is best done in the department of radiology with a radiologist. The commonest post mortem application for CT scanning is in pale-opathology, where historical subjects such as mummies are routinely imaged. Use of CT scanning is however becoming more frequent in larger UK forensic pathology units and on the continent CT facilities are being included in the plans for new specialist forensic mortuaries (personal communication).

#### **Angiography**

Though not widely used, if the expertise and equipment are available there is the potential for the use of angiography in post-mortem practice. Post-mortem angiography has been used in the past in several research applications, for instance to explore coronary artery topography (Fig 6.4).

Locating the source of subarachnoid haemorrhage at post mortem can often be surprisingly difficult. Post-mortem angiography can help to locate ruptured berry aneurysms or vertebro-basilar artery tears in cases of traumatic subarachnoid haemorrhage<sup>4</sup>. For identification of vertebro-basilar artery tears, contrast medium can be injected into the vertebral arteries proximal to their intraspinal course. The technique requires a little practice, because if too much is injected, the contrast can pour out of the defect into the surrounding tissues, obscuring the site of the tear with a "white out". If just a few millilitres are injected at a time bilaterally, a plain x-ray anterior-posterior view of the neck and basal skull can give a perfectly ade-





**Fig. 6.3** A CT scanner being used in a forensic suspicious death case. (a) Complete body. Note still within sealed body bag. (b) Bags containing disrupted human remains. Again, there is no need to open the bags.

quate image. If it is available, real-time imaging of the procedure with fluoroscopy should provide the best results, because obscuring the area of interest with excess contrast can be avoided.

#### **Magnetic Resonance Imaging (MRI)**

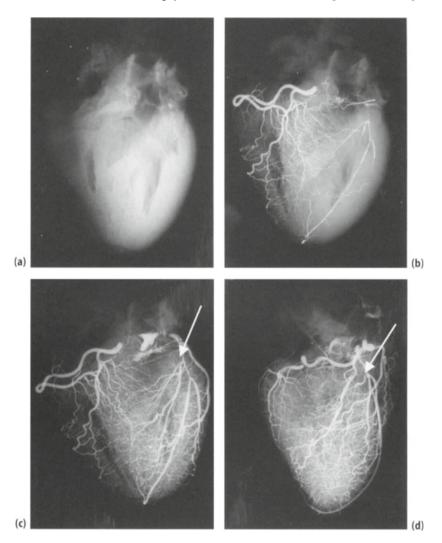
Magnetic resonance imaging has been evaluated as an alternative to paediatric autopsy but as stated above, there is no substitute for a thorough autopsy backed by a full skeletal survey both in terms of cost effectiveness, identification of disease and, most importantly, detection of trauma. Having said this, MRI has been reported to have a role in the demonstration of neurological disease, particularly of the spinal cord which may not be picked up on a "routine" autopsy.

#### Ultrasound

The obvious advantages of x-rays and allied techniques mean that ultrasound has never assumed a practical role in the mortuary. While some work has been done on ultrasonography of neonatal skulls, research involving ultrasound is essentially confined to post-mortem verification of various ante-mortem ultrasonic findings.

### **Practical Considerations**

With any medical test, the doctor must decide if the information to be obtained justifies the time and expense of the investigation. This also applies to the pathologist considering the use of post-mortem imaging techniques. Local issues such as the availability of radiology services will obviously have a bearing on the decision to proceed with imaging, but the physical limitations of buildings and equipment can usually be overcome if the pathologist is prepared to use a little foresight and consideration. The pathologist should be aware of the pressures on the radiology



**Fig. 6.4** The use of angiography to identify coronary artery disease. **(a)** Prior to angiography. Coronary arteries show focal calcification. **(b-d)** Angiography of coronary arteries. Focal areas of stenosis are seen to the vessels (arrows). (Images courtesy of Dr J. Bovee, Department of Pathology, Leids Universitair Medish Centrum, Leiden, The Netherlands).

department, and should appreciate that he or she will get the best from radiographers and radiologists if efforts are made to liaise effectively with them. In terms of priority, radiology of the deceased understandably comes a poor second to the imaging of living patients. To that end, arranging x-rays at a time which does not coincide with busy clinics, filling in the details on a request form fully, and discussing the reasons why an x-ray is needed with radiology staff may mean that the next request for imaging from the mortuary is looked upon more favourably. Remember that in suspicious deaths the radiologist is as much a part of the investigating team as an odontologist, forensic scientist or anthropologist because the final image may be critical to the case, for example where a skeletal survey reveals evidence of child abuse.

## **Health and Safety**

The two major hazards involved in post-mortem imaging are the risk of infection and the risks posed by ionizing radiation.

#### Control of Infection

All the usual measures used to prevent infection in the mortuary should be taken; for instance, wearing of protective clothing and disinfection of equipment<sup>5</sup>. Potentially infective cases can be x-rayed through the body bag, but radiographers may find it easier to get good views with the body out of the bag. Leaving the body in an opened bag with film cassettes beneath the bag may be a reasonable compromise. Depending on the nature of the case, potential DNA contamination by those handling the body may become an issue. DNA contamination can occur even by breathing or speaking over the body. If there are concerns about potential DNA contamination from the investigating team within the mortuary and if appropriate DNA samples have not been taken prior to the arrival of the body at the mortuary, the body bag must remain sealed and an alternative imaging technique, for example fluoroscopy or CT scanning, considered.

#### **Radiation Safety**

In a radiology department it is easier to manage the hazard of ionising radiation, since the department will be purpose built to house the equipment, and the staff should be familiar with both the equipment and local safety procedures. Thus, consideration should be given to taking the body to the radiology department, where more modern equipment will also be available, rather than taking mobile equipment to the body.

When the imaging is carried out in the mortuary, the environment is clearly less controlled and therefore the possibility of unnecessary exposure of personnel to radiation is increased. From the radiographer's point of view, the situation is akin to taking mobile x-rays on the ward. All local rules and procedures regarding mobile radiography should be followed.

A temporary controlled area exists around the x-ray tube and the radiographer needs to be aware of the direction of the x-ray beam. All staff in attendance should comply with the radiographer's instructions in the vicinity of the equipment. If there is permanent equipment housed in the mortuary, then there should be an adequate Quality Assurance programme to ensure it is well maintained. In the UK, radiation safety is covered by the IRMER 2000 document, which states that exposure charts should be available at any control panel to remove the possibility of overexposure due to a lack of familiarity with the equipment<sup>6,7</sup>.

One way of minimising the risks of radiation is to reduce the number of exposures. Always think whether or not the examination is really justified. Careful positioning of the body may take a few minutes longer, but it is usually quicker than repeating the exposure. The more experience the radiographer has, the better the chance of obtaining an adequate image first time. Do not forget what the purpose of the procedure is: the film may be substandard for a radiologist examining a chest

x-ray on a live patient, but if it shows all the foreign bodies in a gunshot victim, do you really need a repeat film? The aim should be to achieve "first time right radiography", reducing the need for repeated exposures. Therefore, it is worth considering whether or not the examination could be carried out in a more controlled environment, which in effect means the radiology department.

To ensure that the highest standards of safety are maintained, careful risk assessment, staff training, maintenance of equipment and adherence to standard operating procedures are all important. But remember that close cooperation between pathologists, radiographers and mortuary staff is the key to safe post-mortem radiography.

#### **Body Handling**

Though radiographers should have had instruction in safe patient handling and lifting, manoeuvring a dead body, with or without rigor mortis, is quite different from handling a live patient. Mortuary staff are, however, more used to handling the deceased, and should be encouraged to assist and advise the radiographer as necessary.

Placing the body in standard radiographic positioning should be the goal. The radiographer will be used to taking x-rays in set "poses" and has a far better chance of achieving adequately penetrated, correctly rotated films if the body is as close as possible to the position that would be used in a live patient<sup>8–10</sup>.

If necessary, rigor mortis can be broken by moving the deceased's limbs to facilitate positioning, but the radiographer should ensure that the pathologist was aware of the position of the body before rigor is broken, since rigor can provide vital information about the position of the body before death. In suspicious deaths, the pathologist must see the body, take all the requisite exhibits for trace evidence and have it photographed for evidential purposes before post-mortem imaging takes place.

#### Clothing

One problem with which radiographers may not be familiar is the presence of potentially dangerous sharp objects on the body or in clothing. These include needles in drug abusers' pockets, and broken glass or fragments of sharp metal in certain accident victims. Furthermore, in suspicious deaths the clothing may contain important trace evidence which needs to be carefully preserved. In these circumstances it is best to photograph and strip the body prior to imaging. This is particularly so in cases where the deceased has been bleeding, as the more the body is moved, the more contaminated the clothing becomes and this will obliterate blood spatter pattern and DNA trace evidence. Finally, stripping the body also allows the radiographer to see anatomical landmarks, which are used to position the body in relation to the x-ray beam.

#### **Radiographic Aids**

Most radiographers will have a "kit bag" filled with radiolucent foam pads, binders, sponges, sand bags, lead rubbers and various other items that are used by local custom or the radiographer's personal preference. In using this kit, radiographers must give due consideration to issues of contamination and infection control.

Though infection control techniques used with the living ought to be equally effective for the deceased, post-mortem work is often unavoidably messy, particularly when dealing with burned or decomposed bodies. It is therefore not surprising that radiographers may be unwilling to use their own kit in these circumstances, let alone x-ray the body. Consequently, it is a good idea to keep a radiographers "kit bag" in the mortuary. Old radiographic aids which have had to be replaced in the radiology department are more than adequate for post-mortem radiography, and having them to hand can save time and improve image quality.

# The Radiographer and the Mortuary

Many of the practical difficulties encountered in post-mortem radiography can be overcome by an experienced radiographer. The problem is that radiographers are often in short supply, and one could forgive them for thinking that taking an extra interest in post-mortem work is an unnecessary burden. This, coupled with the fact that a mortuary can be an intimidating place to the uninitiated, means that few radiographers have expertise specific to post-mortem imaging. Postgraduate courses and degrees are available in forensic radiology for those with an interest.

Those radiographers who do have expertise in mortuary work often find the challenge of producing quality images under difficult and unfamiliar conditions very rewarding. The pathologist in return has an invaluable service which is well worth encouraging. From the pathologist's point of view, it is therefore sensible to promote an interest in mortuary work among radiographers by being approachable, flexible, professional and patient. Patience is particularly important as quality x-rays often take longer than anticipated to produce, but it is still usually quicker to get a good image at the first attempt rather than repeat the procedure. The pathologist should, of course, clearly explain the purpose of the examination, and which views or areas of the body are of specific interest. Once the images have been taken it is easy to forget to provide feedback on the images to the radiographer. Remember that explaining the relevance of the findings in the context of the case only takes a few moments, but is time well spent. The object of the exercise is to ensure that the radiographer feels part of the mortuary team, and to show that their contribution is important and valued.

#### Where to Perform the Imaging

Whether post-mortem imaging takes place in the mortuary or in the radiology department will be determined by a combination of local geography, the availability of staff and equipment.

It is probably easiest to do a skeletal survey of an infant in a radiology department. However, most radiology departments are understandably not keen on having a decomposed body in the middle of a busy clinic. If an x-ray machine is available in the mortuary, then that is probably the best place to carry out the procedure since there will be staff with experience in handling dead bodies available, and it is usually less disruptive and less time consuming in the long run than moving an adult body to the radiology department.

It is worth mentioning at this point that when new mortuaries are planned, or existing facilities modernized, consideration must be given to the need for post-mortem imaging. This includes odontological radiological equipment. It

is important that radiology personnel should be consulted at the earliest stage of planning, so that informed decisions regarding on-site equipment or access for mobile equipment can be made. When building a new mortuary, the inclusion of appropriate radiology facilities may be relatively cheap compared to the overall cost of the project. Furthermore, it is easier and often more cost effective to include them than at a later date. If good facilities are included when a mortuary is built or modernized, it removes the problems associated with transferring bodies to the radiology department and provides modern on-site equipment, with which the radiology staff will be familiar and hopefully more enthusiastic to use.

# **Indications for Post-mortem Imaging**

The key to understanding the indications for post-mortem imaging lies in an appreciation of why vital information may be missed by an autopsy or an imaging procedure. The major limitation of the macroscopic dissection, however diligently performed, is that the pathologist essentially removes tissue layer by layer. He or she therefore only sees the surface of each layer, and despite careful palpation may not detect a lesion just beneath the surface of the tissue. Any pathologist who has searched for a bullet which is clearly visible on an x-ray but proves surprisingly elusive during dissection will be aware of how easy it is to miss a foreign body in a post-mortem examination.

The main limitation of radiology is the presence of artefact, including difficulties with orientation, penetration of x-rays and screening of radio-opaque objects by more dense structures. The radiologist is deprived of the sensory modalities afforded to the pathologist; he or she cannot see colours, use sense of touch to appreciate texture during palpation, or even detect distinctive smells such as alcohol (although many radiologists will be relieved to be deprived of the last!). For instance, a pathologist may see sub-periosteal bleeding around a hairline crack in a rib which the radiologist cannot visualise because of the orientation of the x-ray beam, but may miss callus in a healed posterior rib fracture which is clearly evident to the radiologist.

It follows that dissection during an autopsy and imaging of the cadaver are complementary, and the maximum amount of information will be obtained if information from both techniques is combined.

There are some circumstances where post-mortem radiography should be considered mandatory; these are paediatric autopsies, and any deaths caused by firearms or explosions. Any cases where barotrauma is suspected, such as diving fatalities and cases where there is a possibility of air embolism, should also be x-rayed. There is also an indication for imaging in any case of penetrating injury where a component of the penetrating object may remain in the body, for instance in cases of stabbing where part of the weapon may have broken off in the body or in glassing injuries (Fig 6.5).

There is often a requirement for post-mortem radiology in mass disaster situations, which includes both dental and body x-rays. The need for radiological input will be largely determined by the nature of the incident, in particular the extent of preservation or disruption of the bodies of the disaster victims. In some circumstances, radiology may have a significant role to play in assisting with identification of the deceased, but this may not be the case if most of the victims are suitable for viewing. In some transportation disasters, radiology may provide little infor-



**Fig. 6.5** Broken part of a knife still within the body.

mation into the deaths of individuals that cannot be gained from an autopsy, but may be extremely useful in reconstruction of the event as a whole. Radiological investigations in a temporary mortuary provide specific challenges for both the radiographer and pathologist alike. Health and safety issues are paramount, and close cooperation between pathologists, radiographers and radiologists is imperative if mistakes are to be avoided.

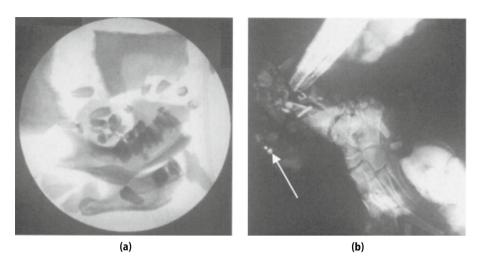
There are other circumstances where post-mortem radiography may not be essential, but may provide invaluable assistance to the pathologist. These include postoperative deaths, particularly if there are allegations of medical negligence. Imaging may be helpful in cases where there may be intracavity gas, for instance if there is suspicion of a pneumothorax. Post-mortem imaging has been used to good effect to detect contraband narcotics in the gastrointestinal tract of dead smugglers.

The pathologist may like to consider post-mortem radiography in any circumstance where the body surface has been compromised, for instance in fire deaths or decomposed bodies recovered from water, particularly if there are any suspicious circumstances surrounding the case (Fig 6.6).

# **Paediatric Autopsies**

A full radiographic skeletal survey must be carried out in all paediatric cases involving infants and younger children<sup>11–20</sup>. Ideally, the films should be checked for adequacy and at least seen by a radiologist (and preferably reported) prior to the post-mortem examination. In any event, a formal report by a radiologist must be available prior to production of the pathologist's final report.

Any lesions identified by the radiographs may direct the pathologist to injuries which could otherwise have gone undetected, particularly healing injuries in long



**Fig. 6.6** (a) The use of fluoroscopy to identify human remains in bags of debris from a fire scene. (b) The identification of personal effects (ear stud (arrow)) within fire debris adjacent to a foot, which may not have been identified due to its size at autopsy.

bones or ribs. It is well recognised that some lesions discovered at autopsy are not identified by radiography, and vice versa (Fig 6.7). Radiographs may fail to reveal rib fractures depending on the angle of the path of the x-ray beam to the injury, and undisplaced fractures will not be obvious to the pathologist on palpation.

The age of the child and the history of the case should always be taken into account when interpreting a skeletal survey. Parents often give a history of accidental trauma, so the pathologist and radiologist must carefully consider whether the circumstances described by carers could account for any injuries.

In the younger infant, the possibility of birth trauma may be raised, so it is important to correlate the radiological and the autopsy findings with the obstetric history. A spiral fracture of a long bone in an ambulant child could have an innocent explanation, whereas such an injury in an infant too young to walk is clearly of great concern. The presence of natural bone disease that can lead to injures that mimic non-accidental trauma must always be considered, for example, osteogenesis imperfecta (Fig 6.8).

Multiple skeletal injuries, particularly if they are of varying ages, are highly suspicious of child abuse. The ageing of skeletal injuries is therefore of prime medicolegal importance, in that the age of a fracture may be used to corroborate or disprove a carer's explanation for an injury. Unfortunately, dating of fractures is fraught with difficulty due to the great variability in the rate of healing between individuals and even in the same person at different times. Therefore, despite the contentions of some experts, ageing of fractures should be approached with extreme caution, and opinions with regard to the possible date of an injury should be expressed only in the most general of terms. The best chance of obtaining a valid estimation of the time frame of a fracture is provided by careful comparison of the radiological findings, the histology of the lesion and, of course, the clinical history. There are some radiographic injuries which are highly suspicious of child abuse, these are given below.



**Fig. 6.7** Fracture to the epiphysis of the humerus neither suspected clinically nor during the external examination at autopsy in a child.

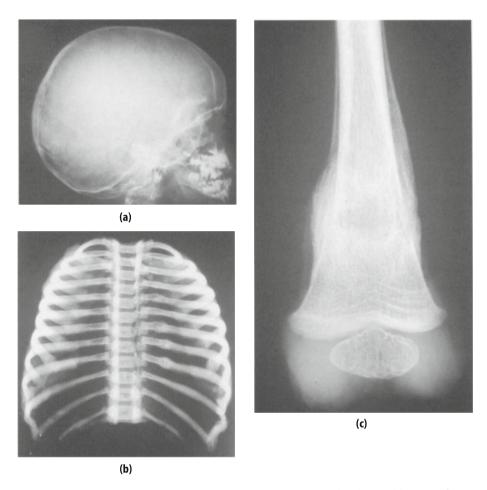
#### Skull Fractures

Accidental skull fractures are generally linear, single and tend to involve the parietal and occipital bones. Comminuted fractures, perhaps involving the base of the skull or frontal bones, are more suspicious of child abuse (Fig 6.9). It may be difficult to differentiate between sutures and fractures on an x-ray, though this should be easily resolved in the course of an autopsy. The pattern of soft-tissue injuries in the scalp may also be helpful in determining the provenance of a skull fracture, and multiple bruises should always be regarded with suspicion.

# Metaphyseal Injuries

Twisting, shaking or pulling by the limbs is a common phenomenon in the abused child. The resultant torsional or linear forces acting on the metaphysis of a long bone may chip or pull pieces of the partially calcified growth plate away from the bone. Metaphyseal and epiphyseal fractures seen on a skeletal survey are strongly suggestive of this kind of abuse. They may appear as chips of bone lying adjacent to the metaphysis. Since the periosteum is easily stripped from the bone in young children, bleeding around the fracture site may lift the periosteum, which subsequently becomes calcified. The calcification under the raised periosteum then appears in the x-ray as a distinctive linear sheath around the bone. Calcification of the bulging periosteum occasionally forms a bridge of calcification between the metaphysis and epiphysis, which is sometimes referred to as a "bucket handle" lesion on x-rays.

Fractures which damage or distort the growth plate may eventually give rise to irregular growth and deformity of the joint.



**Fig. 6.8** A case of osteogenesis imperfecta presenting as a possible non-accidental injury. **(a)** Wormian bones of the lamboid sutures. **(b)** Rib fractures of variable age. **(c)** Long bone fracture (femur).

# Diaphyseal Injuries

Similar periosteal calcification may also affect the shaft of a long bone following direct trauma or torsion, but it may also arise as a result of forceful handling of the limbs during a breech delivery. Thin layers of subperiosteal calcification may also occur simply due to growth of the bone, though in these instances the calcification is symmetrical and limited to the central diaphysis. Irregular unilateral subperiosteal calcification is therefore suggestive of localised trauma. Periosteal calcification takes a week or more to develop, and is therefore indicative of trauma which must have occurred at least a week prior to the child's death.

Spiral fractures of a long bone imply that a twisting force was applied to the bone, and are particularly suspicious injuries in younger infants, as are transverse fractures which are generally caused by force directed at right angles to the long axis of the bone.



Fig. 6.9 Skull fracture to the vertex of the skull in a young child.

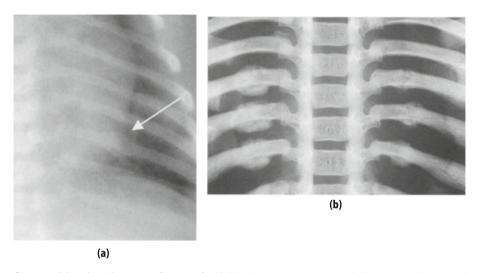
#### **Rib Fractures**

Occasionally, rib fractures which are not seen on a skeletal survey are identified during a post-mortem examination. This may be due to the orientation of the fractures, and does not imply a failure of radiological technique or interpretation. Conversely, radiographs may show fractures which are not immediately evident at autopsy. This is most likely in the case of stable undisplaced fractures or older lesions with callus formation that are situated in a deep position which is not easily palpable. These discrepancies serve as a reminder that effective post-mortem imaging is reliant on close cooperation between radiologists and pathologists, whose roles should be regarded as complementary to one another.

A child's rib cage is more flexible than that of an adult, but can certainly be fractured during unskilled resuscitation. Such fractures are usually anterior or lateral. Posterior fractures adjacent to the necks of the ribs are caused when the ribcage is displaced posteriorly in relation to the spine, which cannot occur if the child is lying on a flat surface<sup>19</sup>. Posterior rib fractures are therefore inconsistent with cardiopulmonary resuscitation. If bilateral they are usually due to squeezing applied by an adult, whereas unilateral fractures suggest more localised trauma. Localised fractures elsewhere imply focal trauma, such as prodding, punching or kicking depending on the age of the child (Fig 6.10).

### **Gunshot Wounds**

It may be surprisingly difficult to locate projectiles in the body of a gunshot victim. The path of a bullet within a body can be extremely unpredictable, and may seem to bear little relation to the expected direction of the gunshot. This in part is due to the fact that most autopsies are performed with the body in the supine position, whereas the position of the body at the time that it was shot may bear no relation



**Fig. 6.10 (a)** Unilateral posterior fracture of a child's rib cage (arrow). **(b)** Multiple posterior fractures of variable age.

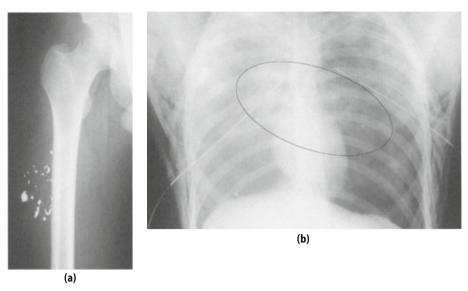
to this. Post-mortem radiography is invaluable in locating projectiles which need to be recovered in as undamaged a condition as possible for examination by ballistics experts<sup>21–25</sup>. Radiographs may also alert the pathologist to potentially hazardous sharp objects, such as jagged edges of bone or metal from a jacketed round.

Projectiles not uncommonly fragment within the body. In these circumstances it is important to recover all the pieces of a projectile, so that markings on the fragments can be matched to the barrel of a weapon. Small pieces of bullet may be very difficult to recover, therefore the x-ray becomes a critical piece of evidence providing a graphic illustration of the distribution of the projectile fragments. (Fig 6.11). Even if there is a single entry wound with a corresponding exit wound, it is still worth performing x-rays, as parts of the bullet may be retained in the body.

A good anterior-posterior view of head and thoracic gunshot wounds on plain x-ray films is usually all that is required, although obviously the areas to be imaged will be determined by both the site and number of wounds. With a single entry wound, this is usually relatively straightforward. However, with multiple gunshot wounds in which some bullets have exited the body it is not always easy to differentiate between entry and exit wounds (Fig 6.12). In these circumstances it is advisable to image the whole body, as bullets may enter the trunk but pass into the limbs.

In a situation where it is difficult to distinguish between entry and exit wounds, the distribution of bone and metal fragments on a radiograph may provide information on the path of a bullet through the body. As discussed above, CT may be able to demonstrate a projectile track in soft tissue. Even with lesser-quality images, it is still possible to deduce the direction of a gunshot if the bullet strikes a bone and then scatters bone fragments along its track before exiting.

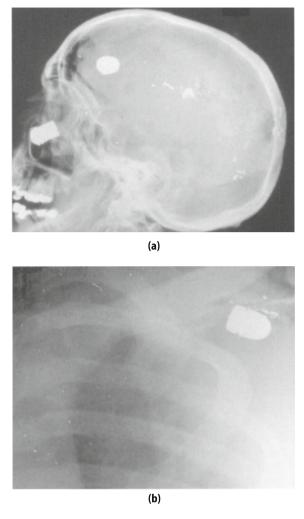
CT scans are often less helpful than plain x-rays, as the extensive shadows cast by the dense metal projectile may obscure fine detail such as bone or projectile fragments, and they may even make it difficult to locate the projectile itself.



**Fig. 6.11** (a) Fragments of a projectile within the right upper leg. (b) Fragments from a projectile within the chest (within marked area). The size of the fragments precludes them been found at autopsy.

Fluoroscopy, although not widely available, is an excellent imaging modality for recovering metal fragments from bodies. The ability to image repeatedly in real time from several angles is ideal for locating projectiles. This technique has been used to great effect in the investigation of multiple fatalities, for instance in the autopsies carried out on mass war graves in the former Republic of Yugoslavia.

Although shotgun wounds are often fairly self-explanatory, x-rays may provide a graphic illustration of the pattern of shot. In a shotgun cartridge, the metal shot is separated from the explosive propellant by wads made of cardboard, felt or plastic. The wads usually provide more information about the cartridge than the pellets, and since identification of the type of ammunition used is extremely important in shotgun wound ballistics, every effort should be made to collect the wads for examination by firearms experts. The wads may be driven into the depths of a shotgun wound in close range shots, but they can be quite difficult to find by dissection alone due to the extent of tissue damage caused by a close-range discharge. Some wads are of a similar density to soft tissues, and may therefore be harder to identify on an x-ray than the much denser metal shot. Occasionally, a shotgun is fired through an intermediate target such as a door or a window, so that fragments of the intervening object may be driven into the wound. Post-mortem imaging can alert the pathologist to the presence of radio-opaque fragments that are of potential forensic significance, but which might be missed during dissection. The x-ray may be used to count the number of projectiles present if there is a question as to the number of discharges of a weapon. It can also identify different sizes of shot, which would support either two types of cartridge fired from one gun, the use of two guns, or two separate incidents with survival of the victim for a period of time following the first injury (Fig 6.13 (a)). Finally, the x-ray should not be confined to the area of the external injury as projectiles may embolise to other areas of the body, indicating that the victim was alive at the time of the discharge of the weapon (Fig 6.13 (b)).

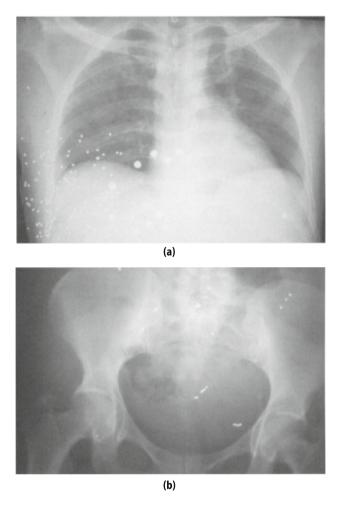


**Fig. 6.12** (a) Projectiles within the skull. (b) Projectiles in the soft tissue of the axilla having passed through the chest.

# Other Penetrating or Sharp Force Injuries

In cases of stabbing, pieces of a knife may occasionally break off in the body. If part of a blade is retained in a wound, it may be used to identify the weapon used in the attack. Furthermore, sharp materials in a wound are a potential hazard to the pathologist during dissection. Therefore, imaging of a wound is advisable if it is suspected that any part of a weapon remains in the body.

Post-mortem imaging may be particularly helpful in glassing injuries, as glass can be difficult to see in the depths of a wound, but it is usually radio-opaque. It is important to obtain as much of the glass as possible for forensic science examination, and as noted above, sharp edges in a wound are a health and safety problem. It is well worth considering x-raying bodies which have been badly damaged in



**Fig. 6.13 (a)** Two sizes of shot present to the chest. The victim had been shot on two separate occasions, surviving the first for several years. **(b)** Gunshot emboli to the pelvis. The principal mass of projectiles was to the chest.

hit-and-run traffic accidents. Fragments of vehicle bodywork can become embedded in the deceased, and if recovered could provide an evidential match to an offending vehicle.

# **Deaths Due to Explosions**

In deaths resulting from explosions, disruption of bodies and burns may complicate recovery of possibly vital trace evidence. The overall pattern of debris and shrapnel in the body can provide valuable information about the orientation of the deceased's body to the source of an explosion. This is particularly helpful to investigators reconstructing an incident if the relative positions of the bodies are known, for instance from a seating manifest in an aircraft.

In terrorist attacks, fragments of an explosive device may well provide vital forensic evidence pointing to the identity of the bombers. Therefore, x-rays are mandatory to locate all the foreign bodies, which can easily be missed by even very thorough dissection due to the extent of tissue damage arising from an explosion.

#### Barotrauma

It can be difficult to reliably demonstrate air within body cavities during a post-mortem examination (Fig 6.14). Post-mortem imaging is a useful method for demonstrating gas within a body cavity prior to an autopsy<sup>26,27</sup>. The exception is in bodies which are beginning to decompose, where microbial action may produce artefactual gas. Pneumothoraces, pneumomediastinum, air embolus within the heart and occasionally pneumocephalus have all been described in post-mortem x-rays. This is particularly helpful in cases of barotrauma, such as underwater diving accidents. It is also advisable to obtain x-rays in those rare medical mishaps where it is suspected that an appreciable volume of air has been introduced into the body. Plain x-rays taken with the body in a decubitus position usually provide the best images for detection of air embolism.

X -rays of the large joints have been used to identify osteonecrosis due to decompression sickness, and to guide sampling of the joints for histology.

# **Neck Injuries**

Most forensic pathology texts contain pictures of radiographs of fractured hyoid bones or thyroid cartilages from cases of strangulation (Fig 6.15). While these may be helpful in illustrating the injuries, they cannot confirm the presence or absence of a vital reaction around the fractures, and consequently they are no substitute for careful dissection of the area. X-raying of the excised anterior neck structures has also been advocated, but again this is no alternative to skilled dissection and photography although it may be used to assess the degree of calcification of the bone and cartilaginous structures.

X-rays of the neck may be helpful in illustrating spinal fractures, for instance atlanto-occipital subluxations, but it is still important to dissect the fracture carefully to exclude post-mortem artefacts. The CT scanner is particularly good at this site, as magnified images in different planes can be used. It may not only show the fracture but also identify underlying natural disease, for example malignancy, which may predispose to a fracture at this site (Fig 6.16).

Post-mortem angiography for the investigation of traumatic vertebro-basilar artery tears has been described above.

#### **Bodies in Poor Condition**

In any circumstance where large areas of the body surface are absent or significantly damaged, the possibility exists of penetrating injuries being missed during a post-mortem examination. The most common cases in which the pathologist is likely to encounter this difficulty are in badly decomposed or burned bodies. Bodies recovered from water are a case in point, since aquatic animals may destroy large areas of skin and subcutaneous tissue. It is therefore worth considering x-raying



**Fig. 6.14** Loops of distended bowel filled with gas in a case of necrotising enterocolitis.

such cases to identify any foreign bodies, particularly if there are any suspicious circumstances surrounding the death.

### Identification of Bodies in Mass Disasters

In any situation where there are large numbers of fatalities, the need to identify individuals is of paramount importance. In mass disasters such as transportation accidents the extent of damage to bodies, particularly by fire, often renders visual identification impractical. In such circumstances bodies are increasingly identified by DNA comparison, although other methods such as comparison of fingerprints or dental records may be used. Post-mortem radiography may, however, provide a rapid and cost-effective method of identification. Items such as orthopaedic prostheses, prosthetic heart valves or pacemakers may have serial numbers which can be used to identify the deceased. X-rays of old skeletal trauma may be compared to ante-mortem films to enable positive identification, or to exclude the body in question as being that of a particular missing person.

A number of comparative x-ray techniques have been used in the past for identification purposes<sup>28–33</sup>. These include matching of the frontal sinuses in skull x-rays, which are said to have a unique pattern in each individual (Fig 6.17). Identification



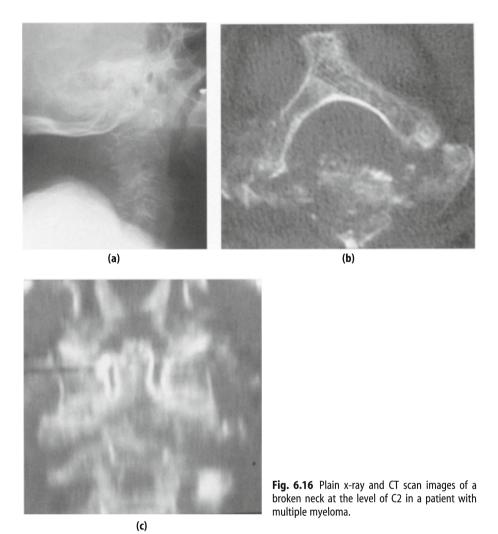
**Fig. 6.15** X-ray of the neck structures in a case of manual strangulation. Fractures are present to the superior horns of the thyroid cartilage.

of bodies with post-mortem x-rays is obviously not possible without the existence of ante-mortem films, and also requires some idea as to the likely identity of the deceased, for instance a passenger manifest. While such constraints also apply to any comparative identification technique, computerized fingerprint and DNA databases tend to give these modalities the edge. However, in many countries such databases are unavailable, for instance in Britain fingerprints will only be on a database if the deceased has a history of involvement with the police, and current law requires that DNA profiles other than those taken from individuals convicted of a crime are destroyed. Therefore, comparison of surgical prostheses or ante-mortem trauma is likely to remain a valid identification technique for the foreseeable future, and postmortem imaging will remain valuable in detecting these features in mass disasters.

The importance of post-mortem radiology in event reconstruction has already been mentioned in the section on explosive deaths above.

### Forensic Odontology

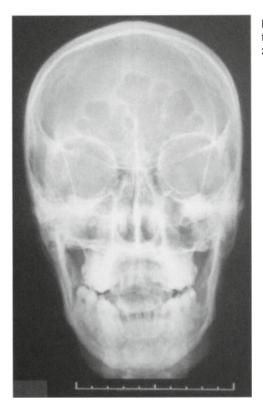
Post-mortem oral x-rays may be useful for comparison with ante-mortem films when the identification of the deceased is unknown, although again one has to have an inclination as to whom it may be for this and other dental comparative work to be of any use. Oral x-rays may be taken with routine plain x-ray machines although optimal films may require the use of specialist dental x-ray machines, which are infrequently found within mortuaries (Fig 6.18). Thus, the machine may have to be



brought to the mortuary, or the jaws removed from the deceased and taken to the machine. The latter technique, however, may not be allowed by H.M.Coroner and therefore this may limit the quality of the images and the extent of the investigation. For those designing new autopsy facilities in the future, the inclusion of dental x-ray facilities would be a very worthwhile addition to the mortuary. For those involved in mass disaster or humanitarian work abroad, dental x ray machines and even fluoroscopes are now manufactured in mobile, compact units which can be carried to the place of work.

### Age Determination

In the case of an unidentified body, x-rays can play a part in age estimation of the remains. X-rays of the epiphyses may be taken and compared to standard charts in an attempt to determine an age range into which the deceased may fall (Fig 6.19).



**Fig. 6.17** Frontal sinus x-ray which can be used for comparative identification if an ante-mortem x-ray exists.



**Fig. 6.18** Dental x-ray machine in use in a mass-disaster mortuary.

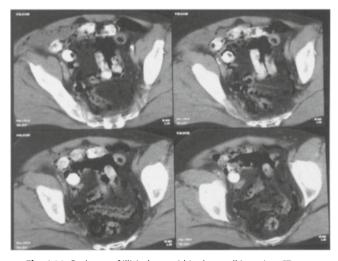




**Fig. 6.19** The use of x-rays to assess epiphyseal fusion.

# Post-operative Deaths and Allegations of Medical Negligence

It is always worth considering post-mortem imaging in perioperative deaths or in cases where there are allegations of medical negligence. This is true even where the deceased has already been imaged prior to death, because x-rays are an excellent method of confirming the anatomical position of medical paraphernalia such as central lines, stents, prostheses, drains and swabs. Obviously, careful autopsy technique is invaluable in determining, for instance, that an endotracheal tube is correctly sited. Nevertheless, if it is suspected that a particular invasive procedure has contributed to death, imaging of the deceased prior to autopsy provides a permanent record which may counter any allegations that the pathologist has inadvertently shifted structures in the course of dissection.



**Fig. 6.20** Packages of illicit drugs within the small intestine. CT scan.

# The Body Packer Syndrome

The body packer syndrome was first described in 1977, and refers to smugglers suffering from drug overdose due to ruptured packages of narcotics concealed in the gastrointestinal tract. The contraband is usually tied in condoms or plastic bags. The packages can be detected by a plain abdominal x-ray, but stool may have a density close to that of the drugs so that they are not always easy to see<sup>34</sup>. Again, the use of CT scan images in such cases not only allows for the identification of foreign bodies within the gastrointestinal tract, but provides information about their location and number (Fig 6.20).

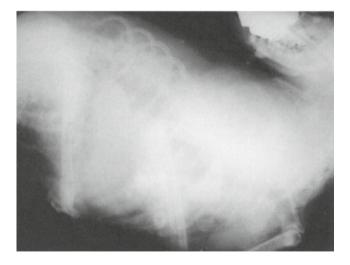
### Poisoning

In cases of suspected heavy-metal poisoning, the metal can form radio-opaque lines in the epiphyses of long bones and within the mucosa of the intestines.

# Deaths Caused by and to Animals

In any situation where the deceased has been savaged by an animal, teeth, claws or other fragments may be left within a wound. Injuries inflicted by domestic pets, usually dogs, may assume medico-legal importance if the owner of the animal is held responsible for the attack. It is therefore worth considering x-raying animal bites, as it is easy to miss a piece of tooth or claw amongst the tissue damage caused by an animal attack, and such evidence may be crucial in identifying the animal concerned.

Domestic pets may be found dead with their owners at scenes which at first may appear suspicious. The classic example is the house fire where the pet may be equally affected as the human and die as a result of carbon monoxide poisoning.



**Fig. 6.21** An x-ray of the burned remains of a dog found in a fire scene where the discovery of the burned remains of the owner were considered suspicious.

If the scene is suspicious then an x-ray examination of the animal should be considered to ensure that it also does not have a projectile within it (Fig 6.21).

# **Post-mortem Imaging in Clinical Audit and Research**

The autopsy should be considered the gold standard in clinical audit, since it is often the only procedure which can incontrovertibly confirm clinical diagnoses and reveal the systemic pathology of the whole body, rather than just the organ system of apparent clinical interest. In other words, it can confirm or exclude whether occult pathology undetected by clinical tests had an impact on the ultimate negative outcome – death. Consequently, post-mortem findings are clearly of interest to all clinicians, and radiologists are no exception. Whether in a formal audit protocol or in the form of informal feedback, the pathologist can be of great help in confirming radiological diagnoses.

Numerous studies have utilised post-mortem imaging, and the possibilities for collaborative research between radiologists and pathologists can only expand with improvements in imaging techniques. The real difficulty in using the autopsy as a research tool is that the numbers of hospital-consent post-mortem examinations is falling year on year, and in England and Wales the coroner's remit specifically excludes research without the express consent of the deceased's next of kin.

To some extent, one could argue that the increased effectiveness of imaging techniques is partially to blame for the decline in hospital-consent autopsies because clinicians often feel they have all the answers from tests performed during life, including scans and x-rays. But the inherent scope for diagnostic error caused by radiological artefacts and the limitations of imaging techniques can never be fully appreciated unless direct visualisation of the body at autopsy remains the clinical audit gold standard.

# The Radiological Autopsy

With advances in medical imaging it has been suggested that whole-body CT or MRI scanning may be able to replace an autopsy, particularly in religious groups opposed to autopsy<sup>35–41</sup>. It could be argued that in a hospital-consent case where a bereaved family has a strong objection to an autopsy, post-mortem imaging may be an option. Whether this is an appropriate use of funds is debatable. Nearly every histopathologist has at some point in his or her career performed an autopsy where the findings disprove a radiological diagnosis. With this in mind, it is clear that imaging without careful dissection in medico-legal cases is not a viable proposition.

# Dos and Don'ts for Successful Post-mortem Imaging

When one is considering whether or not to undertake a radiological investigation as part of the autopsy procedures, always remember a few simple Dos and Don'ts.

#### The Dos:

- DO obtain a skeletal survey on all deaths in infancy and early childhood.
- DO make sure all films are reported by a radiologist before issuing a final postmortem report.

- DO provide feedback to radiographers and radiologists on the findings of the post-mortem examination.
- DO encourage radiographers and radiologists who show an interest in postmortem imaging.
- DO consider post-mortem imaging in cases where the body surface is not intact.

#### The Don'ts:

- DON'T assume that just because a gunshot wound has an entry and an exit wound there is no need to get an x-ray; there may be fragments of bullet or jacket still in the body.
- DON'T forget to carry out the necessary health and safety procedures.
- DON'T rush the radiographer; quality images take time, but getting it right first time is quicker than a repeat procedure.
- DON'T forget to give consideration to the radiology department's clinical commitments.
- DON'T forget to plan imaging facilities with radiology staff when commissioning a new mortuary or refurbishing existing buildings.

### References

- 1. Brogdon BG. Forensic radiology. CRC Press, 1998.
- 2. Evans KT, Knight B. Forensic radiology. London: Blackwell Scientific Publications, 1981.
- 3. Knight B. Forensic pathology, 2nd ed. London: Arnold, 1996.
- 4. Karhunen PJ. Neurosurgical vascular complications associated with aneurysm clips evaluated by post-mortem angiography. Forensic Sci Int 1991;51:13–22.
- Health Services Advisory Committee. Safe working and the prevention of infection in the mortuary and post-mortem room. HSE Books, 1991.
- 6. Department of Health. Ionising radiation regulations. HMSO, 1999.
- 7. Department of Health, Ionising radiation (medical exposure) regulations. HMSO, 2000.
- 8. Bontrager KL. Textbook of radiographic positioning and related anatomy, 3rd ed. Mosby, 1993.
- Bushong SC. Radiologic science for technologists: Physics, biology and protection. 5th ed. Mosby, 1993.
- Swallow RA, Naylor E, editors. Clarke's positioning in radiography, 11th ed. Butterworth Heinemann, 1991.
- 11. Thomsen TK, Elle B, Thomsen JL. Post-mortem radiological examination in infants: evidence of child abuse? Forensic Sci Int 1997;90:223–30.
- 12. Merten DF, Cooperman DR, Thompson GH. Skeletal manifestations of child abuse. In: Reece RM, editor. Child abuse: Medical diagnosis and management. Philadelphia: Lea and Febiger, 1994
- 13. Kleinman PK. Diagnostic imaging of child abuse, Baltimore: Williams and Wilkins, 1987.
- 14. Carty HM. Fractures caused by child abuse. J Bone Joint Surg Br 1993;75:849-57.
- 15 Hobbs CJ. ABC of child abuse. Fractures. BMJ 1989;298:1015-18.
- 16. Nimkin K, Kleinman PK. Imaging of child abuse. Radiol Clin North Am 2001;39:843-64.
- 17. Duncan AW. Radiological features of non-accidental injury. Hosp Med 1999;60:794-9.
- 18. Laor T, Jaramillo D. Metaphyseal abnormalities in children: pathophysiology and radiologic appearance. AJR Am J Roentgenol 1993;161:1029-36.
- 19. Kleinman PK, Schlesinger AE. Mechanical factors associated with posterior rib fractures: laboratory and case studies. Paediatr Radiol 1997;27:87–91.
- 20. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD. Inflicted skeletal injury: a post-mortem radiologic-histopathologic study in 31 infants. Am J Roentgenol 1995;165: 647–50.
- Messmer JM. Fierro AF. Radiologic forensic investigation of fatal gunshot wounds. RadioGraphics 1986;6:4457.
- Di Maio VJM. Gunshot wounds. Practical aspects of firearms, ballistics and forensic techniques. New York: Elsevier, 1985.

- Bixler RP, Ahrens CR, Rossi RP, Thickman D. Bullet identification with radiography. Radiology 1991:178:563.
- 24. Collins KA, Lantz PE. Interpretation of fatal, multiple and existing gunshot wounds by trauma specialists. J Forensic Sci 1994;39:94.
- 25. Hollerman JJ, Fackler ML, Coldwell DM, Ben-Menachem Y. Gunshot wounds: 2. radiology and wound ballistics Am J Roentgenol 1990;155:691–702.
- Ross JAS, Grieve JHK. Underwater diving. In: Mason JK, Purdue BN, editors. The pathology of trauma, 3rd ed. Arnold, 2000.
- 27. Calder IM. Use of post-mortem radiographs for the investigation of underwater and hyperbaric deaths. Undersea Biomed Res 1987;14:113–32.
- 28. Jablonski NG, Shum BS. Identification of unknown human remains by comparison of antemortem and post-mortem radiographs. Forensic Sci Int 1989;42:221–30.
- 29. Rhine S. Radiographic identification by mastoid sinus and arterial pattern, J Forensic Sci 1991;36:272.
- 30. Cuthbert WL, Law FM. Identification by comparison of roentgenogram of nasal accessory sinuses and mastoid processes. J Am Med Assoc 1927;98:1634.
- 31. Murphy WA, Spruill FG, Gantner GE. Radiographic identification of unknown human remains. J Forensic Sci 1980;25:727.
- 32. Asherson N. Identification by frontal sinus prints. London: H.K. Lewis, 1965.
- 33. Messmer JM, Fierro AF. Personal identification by radiographic comparison of vascular groove patterns of the calvarium. Am J Forensic Med Pathol 1986;7:159.
- 34. Karhunen P, Suoranta H, et al. Pitfalls in the diagnosis of drug smuggler's abdomen. J Forensic Sci 1991;36:397–402.
- 35. Myers JC, Okoye MI, Kiple D, Kimmerle EH, Reinhard KJ. Three-dimensional (3-D) imaging in post-mortem examinations: elucidation and identification of cranial and facial fractures in victims of homicide utilizing 3-D computerized imaging imaging reconstruction techniques. Int J Legal Med 1999:113(1):33-7.
- 36. Guidelines on autopsy practice. Report of a working group of the Royal College of Pathologists. The Royal College of Pathologists, 2002.
- 37. Bisset RAL, Thomas NB, Turnbull IW. Post-mortem examination using magnetic resonance imaging. BMJ 2002;324:1423-4.
- 38. Benbow EW, Roberts ISD. The autopsy complete or not complete? Histopathology 2003;42(5): 417–423.
- 39. Roberts ISD, Benbow EW, Bisset R, Jenkins JPR, Lee SH, Reid H, Jackson A. Accuracy of Magnetic Resonance Imaging in determining cause of death: a potential alternative to the autopsy? Histopathology 2003;42(5):424-430.
- 40. Patriguin L, Kassarjian A, O'Brien M. Post-mortem whole-body magnetic resonance imaging as an adjunct to autopsy. J Magn Reson Imaging 2001;13:277–87.
- 41. Bisset RAL, Thomas NB, Turnbull IW. Post-mortem examination using magnetic resonance imaging. BMJ 2002;324:1423-4.

# 7. Body Art and Modification

B. Swift

### Introduction

Body art has been practiced for millennia, the first recorded instances being during the Prehistoric era where natural pigments derived from soils and plant materials were used to decorate the skin. Whether this was performed for spiritual, tribal or purely decorative reasons is unknown, but the act remained popular over subsequent millennia. The methods of decoration have developed into traditions resulting in distinctive cultural identities. Recognition of the forms and methods may not only aid in the identification process of an unknown body but could also provide the investigator with information regarding possible ethnic origins or cultural identity of the deceased. It should be noted, however, that the spread of many such forms through fashion has often blurred these lines of distinction somewhat. Body art may be considered temporary, such as mehndi, semi-permanent, such as body piercing, or permanent, notably tattooing. The appearance at post-mortem examination is therefore dependent on the form of art employed.

Body modification is defined as the act of permanent alteration of body tissues and encompasses both surgical modifications, including therapeutic alterations such as breast reconstruction following mastectomy, and the more cosmetic changes that can be performed outside of hospital, notably ear lobe stretching and even tongue or penile bifurcation. Again, many of these have culture-based histories though for some it may be purely an act of fetishism.

Although the following does not claim to be an exclusive account of all forms or methods it aims to provide the reader with an introduction to both the common and the not-so-common aspects of body decoration which may be encountered during autopsy practice.

# **Body Piercing**

The term, when applied generally, refers to the production of a semi-permanent opening in the skin or a mucosal surface through which an item of jewellery is inserted. Examples of earrings have been uncovered in Sumerian burials dating from 2500 BC though the practice became widespread as evidenced following the examination of grave artefacts from Egypt, Babylonia, China and South America<sup>1</sup>.



**Fig. 7.1** A contemporary example of Mayan sculpture displaying traditional lobar spools.

Despite social prejudice in society historically the practice was not confined to one gender, as eloquently demonstrated by the Assyrian carvings at Nimrud, Iraq (eighth century BC). Despite somewhat altered social judgement today the act of ear piercing remains more acceptable in women even when multiple within a single lobe<sup>2</sup>. Although the same principle applies, the classical lobar piercing is usually excluded from the accepted definition for body piercing, despite such decorations remaining the commonest form of piercing worldwide and throughout recorded history. Less common variations on this theme involve the alteration of the lobe itself with apertures stretched for pendants or the insertion of spools or flares as reflected historically in freezes of Mayan antiquity (Fig. 7.1). The practice was also being performed exclusively by Egyptian women from the Eighth Dynasty though with narrower-gauge plugs<sup>1</sup>. The act, often referred to as scalpelling, continues to be performed today, experiencing something of a renaissance in the West.

The history of non-lobar ear piercings is also well documented with cartilaginous decorations witnessed separately in the Masai tribes-people of Kenya and in the native inhabitants of Indonesia. Whilst lobar piercings have remained popular in Western culture through the centuries, cartilaginous types have only recently entered modern society. As with all piercings, lobar decorations are prone to traumatic avulsion with associated tearing of the pinna (Fig. 7.2).

Contemporary jewellery designs are varied in form though the commonest types are the ball closure rings, the stud and the bar (Fig. 7.3). Ball closure rings are formed from an incomplete loop of metal with a ball placed in the gap. The ends of the loop insert into indents and the ball-piece is thus held in place by tension. Studs refer to a main decoration piece held in place by a backing or "butterfly" piece as often seen used in ear piercings or facial decorations. The bar, be it straight or curvilinear, is passed through an opening and secured in place by an end piece (often in the shape of a ball) screwed into place.

Each piercing possesses a name based either on their anatomical location or a designated name created by the body piercers that popularised the style. Though many have true histories dating back centuries, frequently the names were created to evoke a romantic image with individual false histories devised to elaborate the styles. Separating truth from fiction regarding their use by previous civilisations thus requires a great deal of research<sup>3</sup>.

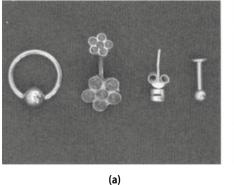


**Fig. 7.2** Common sites of piercings to the pinna: **a)** "Industrial", **b)** Inner Helical, **c)** Helix, **d)** Outer Conch, **e)** "Rook", **f)** "Daith", **g)** Inner Conch (through the cartilage), **h)** Orbital – this refers to the style not the location with a complete loop, half of which sits posterior to the lobe, connected by two piercing holes, **i)** Anti-tragus, **j)** Lobar Spool, **k)** Tragus.

### **Head and Neck Decorations**

Facial piercings historically have focused on the nasal region, with Eastern cultures continuing the tradition of alar piercing, whose origins possibly reflect a supposed submissive female role. It is interesting to note that this type of adornment was also used as a symbol of antiestablishment rebellion in the late 1970s though it has become an accepted art form today, possibly reflecting the creation of a contemporary multicultural society. Special decorative pieces have also been designed specifically for nasal use, being held in situ by a backing screwed into place.

Eyebrow piercing is a relatively modern creation produced using either bars or rings inserted through the subcutaneous tissue and as such being prone to traumatic avulsion or crush-related injuries. Conversely, lower-lip decorations or "labrets" have a long history, having been recorded in both Aztec civilisations and the Inuit Indians<sup>1</sup> (Fig. 7.4). Labrets are a specific decoration still in use today,



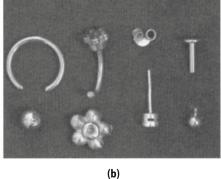
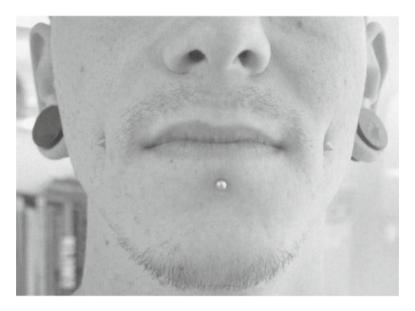


Fig. 7.3 Common forms of jewellery: Ball closure ring, bar, stud and labret. (a) Closed (b) Open.



**Fig. 7.4** Examples of facial piercings (both cheeks and a labret of the lower lip) with lobar spools.

formed by two pieces; an external decoration with a bar passing through the lip and a securing attachment plate within the buccal cavity (Fig. 7.3). Upper lip piercings, often known as the "Madonna", have also become popular, set lateral to the philtrum.

Intra-oral piercings are less common, with gingival, lingual, lingular frenum and even uvular piercings performed<sup>4</sup>. The passing of a bar through the floor of the mouth, secured to the underside of the jaw externally, is a rare variation known as a "sprung". Wearers of lingual jewellery are prone to developing associated dental damage<sup>5,6</sup>. Cracked teeth and fractures have been recorded both in natural and reconstructed teeth following the insertion of jewellery anterior to the lingular frenum. As such, the post-mortem examination of the mouth and pharynx should always be performed with care, preferably with forceps. Fingers should not be placed inside the mouth as a matter of health and safety, thus avoiding potential injuries resulting from contact with sharp dental edges. Such pre-mortem dental change also provides a portal for the entry of bacteria, thus increasing the risk of subacute bacterial endocarditis (See complications later).

### **Chest Wall and Abdomen Decorations**

Nipple piercing has been documented as a fashion accessory since the Victorian era, a period of history otherwise renowned for its morals and prudish sensibilities<sup>3</sup>. The practice is now common amongst both males and females; bars or rings may be used, possibly multiple within the same nipple. Despite commonly held prejudices, the sexual orientation of the individual is not indicated by such decorations nor does it necessarily preclude subsequent breast feeding<sup>3</sup>.

The Madison is a rarer form of piercing localised above the suprasternal notch

at the base of the neck and usually in the form of one or two bars placed through the subcutaneous tissue.

Navel decorations (usually sited superiorly) possess no cultural significance, being created purely as a fashion statement that gained widespread popularity in the mid-1990s; it should also be noted that its popularity extends across many age ranges and social classes<sup>7</sup>.

### **Genital Decorations (Male and Female)**

Genital decoration is regarded by modern society as an extreme form of decoration though the Dayak tribesmen of Borneo have traditionally performed such decoration. A bone shard is passed through the glans penis as either an Apadravya (ventrodorsal piercing through the glans) or an Ampallang style (coronally through the glans). The act may have been to inhibit sexual relations as well as being a decorative form. Vatsyayana also described genital piercings in his writings "The Kama Sutra" (circa 300 AD). As illustrated within the text, genital jewellery is used not only as a decorative form but also as a means of sexual stimulation often for both partners. Today, genital piercing, especially female, continues to be requested as a means of increased sexual arousal<sup>8</sup>. As with nipple piercing, no assumptions regarding sexual orientation should be drawn, being common amongst both heterosexuals and homosexuals alike.

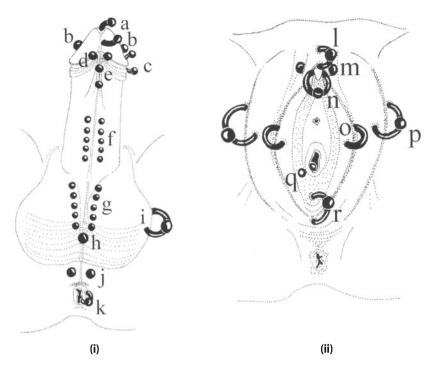
The commonest form of male genital piercing remains the Prince Albert, or P.A., the vague history of which suggests it was used in the Victorian period to secure the penis to one side thus preventing an "indecent" appearance when wearing fashionably tight trousers of the era<sup>9</sup> (Fig. 7.5(i)). Its eponymous origins, however, remain unsubstantiated. The ring is passed into the urethral meatus and out through a communicating opening created along the ventral aspect of the penis. (A reverse Prince Albert passes similarly from the meatus and exits to the dorsal aspect of the glans.)

Female genital piercing is also not uncommon, often performed around the clitoris to enhance sexual stimulation<sup>8</sup>, though current legislation within the UK actually renders it an illegal act under the "Prohibition of Female Circumcision Act" (Fig. 7.5(ii)). Similarly, the decoration is included under the World Health Organisation description of genital mutilation<sup>11</sup>. Despite this, to the author's knowledge, no legal action has been taken against body piercers in the UK.

Essentially, almost any part of the body can and probably has been pierced including the inter-digital webs of the hand, through the hand itself and even long subclavicular piercings by an extended bar<sup>4</sup>. The possible complications caused by the creation of the latter are extremely worrying in themselves.

# **Complications of Body Piercing**

The complications associated with body piercing have been extensively documented, focusing generally upon the localised conditions<sup>9</sup>. It should be reiterated, though, that potentially fatal incidents have occurred: two cases of subacute bacterial endocarditis following body piercing have been recorded in the literature<sup>12,13</sup>. Although one of the patients possessed a documented congenital cardiac valve anomaly<sup>13</sup>, the absence of identifiable risk factors in the second individual should be cause for concern. Indirectly, oral piercing may also constitute an increased risk through the longer-term complications of secondary dental damage, as described



**Fig. 7.5** Genital piercings. (i) The male. (ii) The female: a) Prince Albert, b) Ampallang, c) Dydoe, d) Frenum, e) Dolphin, f) Shaft Ladder, g) Scrotal Ladder, h) Trans-scrotal, i) Hafadas, j) Guiche, k) Anal, l) Hood, m) Triangle, n) Clitoral, o) Inner Labial, p) Outer Labial, q) Hymen, r) Fourchette.

above, as well as the possible primary introduction of bacteria at the time of piercing. The problem may be confounded by the lack of awareness on behalf of those performing the act as to the need for antibiotic cover in patients either possessing such anatomical risk factors or receiving oral decorations<sup>14</sup>. As well as a risk of hepatitis viral infections spread through body piercing, a single case of possible HIV infection has also been documented<sup>15</sup>. The possibility of a blood-borne viral infection should be considered at autopsy.

# **Removing Jewellery**

The removal of jewellery items may be necessary at autopsy for one of several reasons:

- 1. The jewellery itself may assist in the identification of an individual, especially in cases of advanced putrefaction or exposure to extreme heat, thus constituting evidence.
- 2. The next of kin may request the return of all jewellery and as such each item should be removed following the undressing of a body with preferably no unnecessary damage to the body.

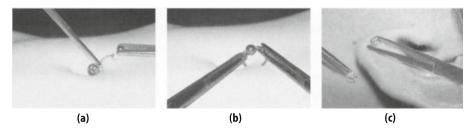


Fig. 7.6 Methods of safe removal of (a) a stud, (b) a ball capture ring and (c) a bar, as described.

- 3. The jewellery may prevent adequate radiological views. Odontoid peg views in suspected cervical spine fractures may be obscured by lingular jewellery<sup>16</sup>. Alternatively, should the department possess access to computerised tomography facilities, the x-rays may be "scattered" by the presence of metal, producing artefactual shadowing thus necessitating its removal to improve the quality of views.
- 4. And, as with all forms of jewellery, it should be photographed or documented in writing to avoid the accusation that it has been removed and "stolen" in the mortuary.

The pathologist, therefore, should be aware of the methods for safe removal of such objects (Fig. 7.6): The stud is removed simply by the detachment of the butterfly piece posteriorly allowing extraction of the remaining section. This is best performed with artery clamps to avoid accidental injury. Ball closure rings require the removal of the holding tension. Two artery clamps or rat-toothed forceps holding the loop should be used to pull in opposing directions to release the holding ball. Care should be taken not to lose the ball and as such it should be released into a catching pot or on to a piece of gauze. The removal of a bar is completed by the unscrewing of one end by holding the bar with one artery clamp and unscrewing an end with a second artery clamp.

Alternatively, it is important to respect the beliefs of certain faiths. As such, all jewellery, including sacred threads and kara (steel bracelets), should remain in place on the body of a follower of Sikhism<sup>17</sup>.

### **Alternative Piercing Forms**

"Pocketing" or "stapling" is a form of body art, the process involving a long bar of metal either end of which is inserted sub-dermally with the metal between remaining visible lying across the skin surface. The visual effect is like that of a large staple (hence the name) and is often performed around the arm or leg areas.

Play piercing, however, differs to that of the definition of body decoration as detailed above. The act is purely the insertion of a sharp object such as a hypodermic needle, pin or nail into the skin without the subsequent placement of a decorative piece. The piercing ritual and the seeking of the pain associated with the act provides the reason for such activities and may form part of a generalised consenting sadomasochistic experience. Such acts may be focussed around the face, arms, breasts or genitals, and is often self-inflicted. The resultant bleeding and bruising could be misinterpreted as the stigmata of intravenous drug abuse, though

the distribution being unassociated with an identifiable vascular structure should preclude such a misdiagnosis. In extreme forms, the injuries could falsely resemble the evidence of torture.

# The Body as a Canvas

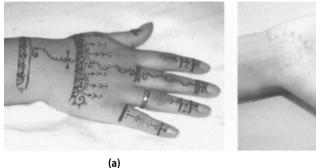
The creation of cosmetics derived from prehistoric acts of body painting has been recorded extensively in the Egyptian era amongst both males and females. Though the constituents have altered over the centuries, the main focus of application remains similar, being around the eyes, cheeks and the lips, possibly accentuating sexually attractive features. Though females mainly use such decorations today, some males often use make-up, be it to highlight features such as eyelashes or even as part of deliberately emulating the female. Like many art forms, certain cosmetics have also developed from religious origins.

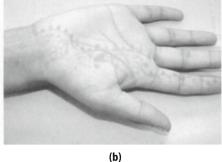
# Bhindi, Tilaka, Bindiya, Kumkum or Sindhura

The Tilaka is a religious mark applied centrally to the forehead above the eyebrow line by the followers of Hinduism. The mark is usually circular, being vermillion in colour, though different Hindu sects have recognisable variations. Followers of Shiva traditionally apply three horizontal bands of ash whilst worshippers of Vishnu use either saffron or crimson pigment painted into a U-shape. The continued practice may reflect the mark formed historically during marriage by the groom who applied his own blood to the forehead of his bride.

### **Mehndi or Henna Tattoos**

The Mughals introduced mehndi to India and Pakistan from North Africa and the Middle East in the twelfth century where it gained popularity amongst the Rajputs of Mewar in Rajasthan. Over time, mehndi gained cultural significance and became an essential part of auspicious Muslim, Hindu or Sikh occasions, particularly weddings. The powder formed from the crushed leaves of the henna plant, (Lawsonia inermis), is mixed with water, oil and spices. The resultant mixture is then piped on to the skin, notably the hands and feet, to produce intricate designs. The pigment, though not permanent, is retained on the skin surface for several weeks. As such, even when fading, the designs may still be recognisable at post-mortem (Fig. 7.7). Equally, fashion has resulted in the spread of the art to the West with the creation of cheaper commercial forms of the dye. The outcome of this includes reported incidents of localised allergic reactions<sup>18</sup>. The application of henna preparations to the skin is also potentially lethal, notably for infants with inborn errors of metabolism<sup>19</sup>, and, although no cases have been reported to date, the use of walnut powder in certain commercial henna preparations would constitute an anaphylactic risk for persons sensitised to nuts. Any suspicion of such would require appropriate post-mortem biochemical and serological investigation.





**Fig. 7.7** Henna tattoo. (a) Recently applied, with defined raised edges to the pigment mixture, and (b) one week post-application, resulting in a faded discolouration to the skin surface.

# **Tattooing**

Tattoos, in the general sense, refer to the creation of permanent pigmentation due to the insertion of non-native pigments into the dermis, though it should be noted that Indian Ink tattoos are created endoscopically within the gastrointestinal tract mucosa to identify areas of pathological interest<sup>21</sup>.

Tattoos may be either intentional or non-intentional. The latter constitutes the effects of lifestyle or employment, notably the carbon dust that becomes entrapped within cutaneous wounds of coal miners<sup>22</sup>. Despite the closure of the majority of working mines within the UK, the lesions persist as an indicator of prior employment especially over the forehead, cheeks, hands and knees of ex-coalface workers. As such, these stigmata may indicate an increased risk of associated industrial illnesses such as chronic obstructive pulmonary disease or pneumoconiosis.

Amalgam tattoos frequently form on gingival surfaces adjacent to areas of dental restoration and remain recognisable even in the absence of the repaired tooth, or as areas of radio-opacity on dental radiological investigation<sup>23</sup>.

Non-intentional tattoos also include the residue deposited following the close-range discharge of ballistics and explosives. Though mass spectrometry may assist in the confirmation of the components, a routinely available histochemical stain will provide relatively rapid confirmation of metal fragments within histological sections of skin<sup>24</sup>. In the case of gunshot injuries, this tattooing may not only assist in the estimation of the distance of discharge from the body, but may also assist in the identification of the entrance wound on the body (Fig. 7.8).

Intentional tattoos can be further subdivided into artistic (being either professional or non-professional) and therapeutic. The latter are produced during the treatment of malignancies simply by the injection of ink or a charcoal suspension into the dermis to produce a reference point for future courses of focussed beam radiotherapy. In the absence of clinical records these iatrogenic markers remain to provide the pathologist with valuable background information. Areolar reconstruction following mastectomy can also be performed through tattooing to produce an aesthetically realistic appearance<sup>25</sup> and tattoos have also been used to conceal vascular malformations.



**Fig. 7.8** Gunshot discharge tattooing around the right nostril. The firearm had been discharged such that the projectile had entered the head via the nostril orifice.

Artistic tattoos are a decorative form, one with a history dating back several millennia, as witnessed by the marks present on Egyptian mummies and the alpine iceman, affectionately known as "Oetzae"26. The practice of tattooing has been geographically widespread throughout its history. The males of the Marquesa Islands were fully tattooed from adolescence as a display of bravery, wealth and sexual attractiveness. Even the Britons were noted for their body art. Caesar, visiting England as part of an invasion army, commented on the Picts, named after the tattoos or "pictures" decorating their bodies. Created with natural pigments, notably woad (Insatis tinctoria), the art remained popular until the Roman Empire converted to Christianity in 325 AD<sup>27</sup>. Passages of the Old Testament were interpreted as forbidding the act of tattooing ("Ye shall not make cuttings in your flesh for the dead, nor print any marks upon you; I am the Lord" Leviticus 19:28). Christians historically regard their bodies as a temple or vessel for their God and altering it in such a manner is therefore interpreted as defilement. Pope Hadrian therefore formally banned tattoos in 787 AD. Despite such attempts to prevent the act, some regions actively displayed their faith by the use of such means as recorded in Catholics of the Balkans. The art form diminished in popularity over the centuries until Joseph Banks, a member of Captain Cook's Endeavour explorations, published his accounts of the Tahitian population in 1769. The visitors adopted the decoration and its name, "tatau", with Bank's accounts and illustrations facilitating its spread.

Although there has been an increased acceptance of professional tattoos, several anatomical sites remain taboo, with facial decorations continuing to be judged

harshly by society. These emotive responses have changed little since the days of Herman Melville, who applied this form of judgement descriptively within the facial tattoos of Queequeg to instil a feeling of initial unease within the narrator, Ishmael, and hence the reader. Maoris, like Queequeg, used these distinctive designs as part of the Ta Moko, a spiritual, mental and physical experience during which the skin is "chiselled" and ink inserted (Fig. 7.9). The act is performed by a tohunga (priest and scholar) and, although regarded as war paint by outsiders, the designs are specific, acting as personal spiritual emblems to protect them. Their designs were part of their cultural heritage; however, as a result of the Western-based judgements regarding facial tattoos, there has been a diminished incidence of Ta Moko in New Zealand and as a result one of the cultural identities and traditions of the Maori may be lost forever<sup>28</sup>.

Artistic tattoos may be described as either professional or non-professional, the former often involving more than one colour forming distinct patterns or designs. The latter is "home-made" with a sharp instrument, such as a sewing needle, a knife or pen, and writing ink, charcoal or ash forming simple designs or words usually in a single colour only<sup>29</sup>. These are often created in institutions performed either by the individual or a friend/inmate. The most notorious form historically was the creation of a permanent identifying number tattooed onto the forearm of those imprisoned within the concentration camps of the Second World War. Elderly survivors of such camps may still possess these tattoos.

The suboptimal hygiene associated with tattooing within prisons causes an increased risk of blood-borne infection, although the real reason for the increased



**Fig. 7.9** An example of "Moko" – traditional Maori facial tattoos, usually involving the chin and mouth area. (Muir and Moodie Dunedia Collection, 1902).

prevalence of such infection may be the widespread use of intravenous drugs amongst inmates<sup>30</sup>. Cases of both Hepatitis B and Hepatitis C infection and even a case of *Mycobacterium tuberculosis* inoculation have been reported resulting from such tattooing under poor hygienic conditions and as such, the taking of increased precautions should be considered during autopsies on individuals bearing these marks<sup>31,32</sup>.

Professional tattoos are created generally within licensed premises, first through the creation of a template drawn on the site to be decorated (Fig. 7.10). The lines are then illustrated permanently through the use of a tattoo machine bearing multiple needles with a vibration cycle of between 50 and 3,000 movements per minute<sup>29</sup>. The oscillating needles puncture the skin to a depth of 1–2 mm, depositing ink particles within the papillary and reticular dermis<sup>33</sup>. If a small area is illustrated to too great a degree, the epidermis may become torn and macerated, ultimately loosing the pigmentation. The result is a tattoo that, in places, shows areas of fibrosis and scarring; a sign of low artistic experience<sup>33</sup> (Fig. 7.11).

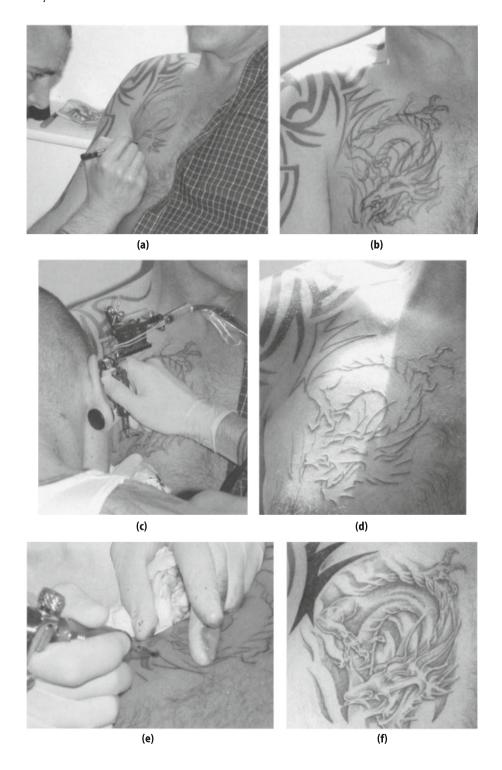
The presence of tattoos at post-mortem examination has long been established as a means of identification. Occasionally, personal information may be inferred from tattoos, such as the intra-oral mucosal tattoos associated with drug-dealers or users<sup>34</sup>. As such, the location and style of tattoos should be documented with the possibility of photographs being considered in cases of unconfirmed identity. However, many tattooists possess a limited repertoire from which clients may choose, with identical designs used by many separate establishments (Fig. 7.12). Truly unique tattoos are less common though where recognisable the manner of design may be of assistance. Affiliation or membership of certain groups, notably gangs, paramilitary organisations or specialist armed forces, have been witnessed by the author, as represented by specific tattoos, all of which may aid in the interpretation of the events surrounding the individual's life and death.

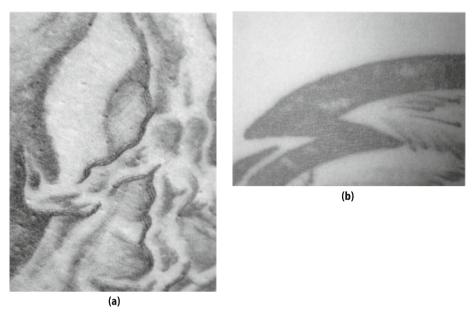
Truly cosmetic tattooing has also been an option to create a permanent eye-liner effect, thus no longer requiring the wearer to continually apply make-up on a daily basis<sup>35</sup>. Recent advances in ink technology have also produced a fashion for fluorescent pigments designed to illuminate under ultra-violet lights commonly used in nightclubs. A portable ultra-violet lamp in the mortuary may assist in the recognition of these and also aid in the delineation of designs in less-preserved bodies, where the stratum corneum has sloughed off<sup>22</sup>. Such equipment, however, must not be used prior to sampling of the body for DNA trace evidence as, depending upon the frequency of the lamp, the DNA may be denatured and thus critical evidence lost.

Tattoos may be intentionally removed by dermal abrasion or laser treatment, leaving an acceptable scar or residual pigment<sup>36</sup>. Larger pieces of body art may also be used to disguise older tattoos that have become no longer acceptable to the owner. Residual areas of the original work can often be identified in such cases.

The creation of tattoos may result in localised reactions and can produce lymph node swellings mimicking neoplastic disease<sup>37,38</sup> (Fig. 7.13). Systemic complications have long been recognised, the first being the documented outbreaks of extragenital syphilis reported in the late 1800s<sup>39</sup>. Cases of systemic sarcoidosis activation within a tattoo and primary cutaneous malignancies arising in designs have since

**Fig. 7.10** The process of creating a "professional" tattoo. (a) Hand-drawing the design outline. (b) The drawn design, to integrate with the existing decorations. (c) Creation of the tattoo outline (d) The completed outline. (e) Shading produced with a diluted pigment suspension. (f) Finished design.





**Fig. 7.11** Close up of **(a)** a good-quality professional tattoo and, **(b)** a poor-quality professional tattoo showing areas of scarring, referred to by tattoo artists as "holidays".

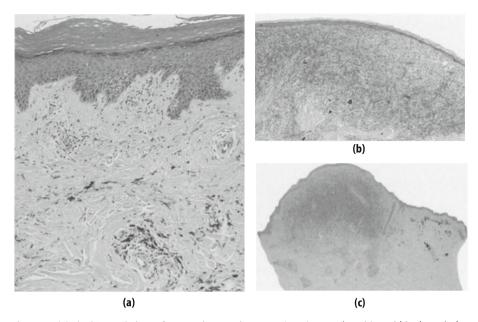
also been published<sup>40–43</sup>, though the risks of viral diseases appear to be less than those associated with non-professional designs. Although the hazard of Human Immunodeficiency Virus (HIV) transmission is often quoted, little data exist in the literature to back this statement. The subject of transmissible prion disease resulting from tattooing or body piercing has also yet to be formally addressed within the scientific literature. The prion protein requires higher temperatures and longer sterilisation times than viruses or bacteria and, as such, viable material could, theoretically, remain on re-useable instruments.







Fig. 7.12 Examples of popular tattoo styles commonly encountered.



**Fig. 7.13** (a) The histopathology of a normal tattoo demonstrating pigment deposition within the reticular and papillary dermis, and within macrophages producing perivascular infiltrates. (b) A prominent lichenoid reaction to tattoo pigment, which may still be recognisable in the background dermis at low power. (c) A pseudo-lymphomatous reaction to tattoo pigment. Similar dense inflammatory reactions may also be seen in regional lymph nodes, mimicking a lympho-proliferative malignancy. (Photographs courtesy of Dr. G. Saldanha, University of Leicester, UK.)

# **Scarification and Branding**

Historically, tattooing was found in all corners of the discovered land except for the sub-Saharan region of the African continent. With naturally pigmented skin, tattoos would not be as visible. As a result, the art of scarification developed, being the deliberate creation of scars, notably keloid in nature, to produce a design or pattern. The act has been revived in the West, being of higher prevalence amongst college students. Modern methods of creation usually involve the use of a tattoo machine lacking in pigment. The repeated puncturing of the dermis produces an inflammatory reaction that ultimately results in the required fibrosis.

Branding has also experienced a resurgence, again mainly with younger people<sup>44</sup>. Originally used to assert ownership over livestock, the hot iron is also used on human skin, though lasers are being used in the more professional establishments. Reportedly painless, the use of the branding can show allegiance of that person to a social group or gang.

Both acts are intentional and used to create definite patterns, words or images. As such, these will often differ from the appearances of deliberate self-harm or the cutting of suicidal intent, which are either in areas hidden by clothing or focussed over vital structures. Fewer professional establishments offer these services therefore making identification of the creator easier.

# **Body Modification**

Body modification has an extensive history relating to both fashion and social ideals that often vary across countries, an extreme example being foot binding, used to create smaller deformed feet and now outlawed in China. Such modifications, seemingly bizarre to the West, were considered to be a more attractive attribute. Conversely, the vogue for wearing corsets by both sexes in the Victorian era to create a fashionable appearance or posture was equally regarded by Asians as unusual. Methods currently in practice utilise both these non-surgical techniques but also surgical means to alter the external appearance of the body.

# **Non-surgical Modifications**

The manner in which hair is worn may be in response to current trends or in keeping with religious doctrine. The Torah dictates that the hair of Orthodox Jews should not be cut round the corners of ones head nor the corners of the beard. Rastafarians also grow their head hair to form dreadlocks, representing the Lion of Judah. Removal is forbidden and, as a mark of respect, care should be taken to preserve the dreadlocks during examination of the scalp and removal of the brain. Sikhism also forbids the removal of any body hair (known as the "kesh") as a symbol of holiness and strength. It also marks an acceptance of a simplistic life, denial of pride and a willingness to accept God's gift. The hair is kept in a knot, covered with fabric, until the male reaches maturity after which a turban may be worn. Again, as a mark of respect, the hair of the deceased should be left as intact as possible.

Hair itself can provide information such as toxicology, race determination and DNA analysis<sup>34</sup>. It is recommended that hair be measured at three points during a forensic post-mortem, with any patterns of balding, artificial hair colouration or the presence of any hair extensions or wigs being noted.

Iris colouration or pupil shape may also be altered through the use of contact lenses, creating a non-natural colour or an appearance such as that of an animal's pupil. Care should be taken in examining the eyes at necropsy to ensure correct documentation of natural eye colour and whether in fact the eye present is real or a prosthetic.

Fingernail extensions, manicuring and the painting of elaborate designs on nails have increased in popularity, emulating fashions originating in the United States. Beauticians or manicurists may be able to recognise their own work, aiding identification issues. Once photographed and fingerprinted, nail varnish should then be removed to examine for uncommon stigmata of natural diseases such as the splinter haemorrhages of subacute bacterial endocarditis or the Beau's lines of chronic disease to assist in pinpointing a natural cause of death.

The natural hyperkinetic facial lines that develop with age can also be evened out through the use of dermabrasion (commonly used to treat the scars of acne), with the option of chemical "peel" solutions such as a 20% glycolic acid solution, via pulsed vaporisation lasers, or through the use of Botulinum toxin injections<sup>45–49</sup>. Despite the toxic effects of the latter, surprisingly few side effects have been recorded.

# **Surgical Modifications**

Surgical modification of tissues is a more extreme form of body modification in which any procedure is used to either alter or resect tissue, or to enhance natural

tissue artificially. The form may be therapeutic or cosmetic, the latter often being performed in private clinics though in certain circumstances it is even possible to do in the home environment.

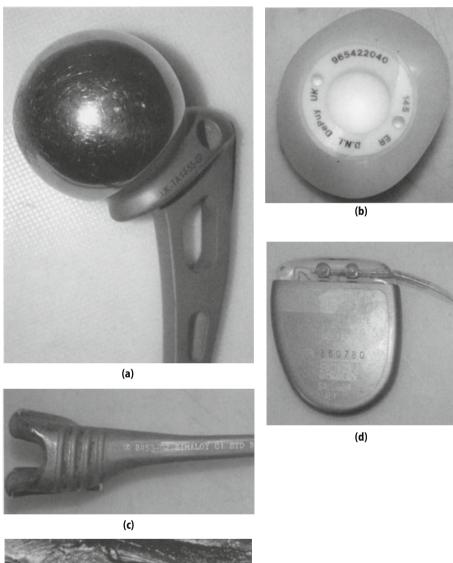
Dental alterations, it could be argued, are a form of body modification used to create a socially ideal appearance. This may include restoration work, realignment treatment, crowns or caps. Dental comparison with pre-mortem records could assist with identification, though examiners should be aware that prostheses may even be marked with the name of the owner, albeit infrequently. Even following exposure to adverse temperatures it may be possible to recognise such work owing to the lips and tongue acting as a protective barrier, shielding any braces or dentures from heat exposure. Consultation with a registered forensic dentist is strongly recommended in such cases, where root canal enlargement or fragments of residual amalgam may be recognisable even in cremated remains<sup>23</sup>.

Though precious-metal caps have been used functionally for many years, gold may be used as a form of fashion, possibly with the insertion of precious or semi-precious stones into the surface of a tooth. More extreme forms of dental alteration involve the "filing" of teeth into shapes, usually pointed, to produce forms reflecting animal canines (Fig. 7.14). The use of such dental alterations may be tribal in origin, such as the Hindu festival of "Potong gigi" celebrated in Bali as a coming of age ritual amongst adolescents, though the origins of dental filing also lie in tribes in which cannibalism formed a part of their society. The followers of supposed vampire mythology also use these modern applications with a greater frequency today. A case of a suspicious death of an adult female with filed teeth and a quantity of human blood within the stomach at autopsy is known to the editor of this book (personal communication).

Therapeutic modifications would include the use of internal surgical prostheses, such as a hemi-arthroplasty, plating and screwing for fixation of bones, or even the use of full external prosthetic limbs (Fig. 7.15). The latter are often created uniquely for the individual based on the shape of the distal limb portion remaining. Such



**Fig. 7.14** An example of teeth filing to produce an animal-like appearance. (Image courtesy of Eric Sprague (Photo by Allen Falkner)).





(e)

**Fig. 7.15** Examples of prostheses, each with unique identifying numbers. **(a)** Femoral head. **(b)** Acetabular prosthesis. **(c)** Knee replacement. **(d)** Cardiac pacemaker **(e)** Surgical plate and screws in situ aiding identification of a mummified female corpse.

uniqueness may assist identification. Similarly, although mass produced and not unique, surgical implants such as a hemi-arthroplasty are individually numbered. Cross-reference between the implant and the recorded information detailed in the surgical notes of an individual will allow identification. This applies to both large prostheses, such as joints, and smaller implants such as an artificial lens.

Cosmetic alteration is used to produce an acceptable appearance to that individual, or one they believe would be pleasing to others, and as such frequently involves areas "on display", notably the face, neck or chest areas. As will be discussed, the individual's ideals often vary greatly across societies resulting in a diversity of modifications.

Prominent earlobes are frequently altered in childhood to change the angulation of the pinna leaving a vertical scar posteriorly where skin or cartilage has been resected. Rhinoplasties are also not uncommon and are usually created at the request of the individual. Face lifts (rhytidectomies) can alter facial lines and create a more youthful appearance. Such procedures, like the previously mentioned non-surgical methods, could affect subjective age estimations made at post-mortem.

Liposuction aims at resculpturing body lines and is one of the most commonly requested cosmetic surgical procedures performed in the United States. The operation requires small incisions to be made, usually on the abdomen, the legs or the neck, through which a wide-bore cannula is passed under local anaesthesia with sedation or general anaesthesia. Tumescent liposuction, being the commonest form, requires the infusion of several litres of dilute adrenaline and lidocaine into the subcutaneous tissue prior to the removal of the adipose through means of suction. The risks of such a procedure are increased if the patient requires general anaesthesia, however, liposuction is not without risks itself. Cases reported in the literature include pulmonary oedema with cardiac failure induced by the infusion of large volumes of fluid, and necrotizing fasciitis<sup>50,51</sup>. The potentially traumatic nature of the procedure has been known to result in a large retroperitoneal haematoma following bilateral lumbar artery transection<sup>52</sup> and a case of multiple small-intestinal perforations has also been reported<sup>53</sup>. Deaths commonly ascribed to liposuction have been as a result of pulmonary emboli, the aforementioned pulmonary oedema, lidocaine toxicity and cases of fat embolization confirmed by appropriate histological means on fixed lung sections.54-58 As such, any post-mortem performed upon an individual who recently underwent such an operation should include appropriate toxicology and assessment for both thrombotic and fat emboli.

Though some private clinics offer a selection of alterations created under sterile conditions, many seemingly minor surgical amendments are created in the home environment. One such form that has gained popularity is the stretching of the pendulous aspect of the ear lobes. The origins of this act lie in ancient populations, produced by the "scalpelling" of an opening in the pinna and the insertion of either a wedge or a spool, as mentioned previously. Alternatively, the attachment of a heavy pendant elongates the skin further over a long duration.

An individual may even wish to possess a bifurcated tongue producing a "forked" appearance that is aesthetically pleasing to that person and doesn't interfere with eating or talking (Fig. 7.16). Again, this is commonly performed in the domestic environment with a scalpel and dental floss.<sup>4</sup>

The need for implanted prosthesis into the breast can be divided into three main requirements: The first is to enhance the natural female form, the second to create acceptable breasts in females, possibly to compensate for previous mastectomy or to conceal mammary aplasia especially when unilateral, and the third being the



**Fig. 7.16** Bifurcation of the tongue. The individual reports no loss of mobility, being able to move each half independently of each other. (Image courtesy of Eric Sprague (Photo by Allen Falkner.))

creation of breasts during gender-reassignment operations. The types and contents of implants vary, some being prone to leakage resulting in an associated stromal reaction to the contents with fibrosis surrounding the implant remnants (Fig. 7.17). Conversely, scars of breast reduction are seen, being elliptical along the inferior surface of the breast usually with conservation of the nipple and areola. Pectoral implants to alter the male chest are also increasing in popularity.

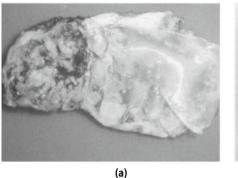
#### Genitalia

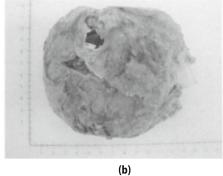
The deliberate alteration of the genitalia may be seen in either sex. Most are consenting, especially amongst adult males and females who wish to modify their sexual appearance, though unconsented acts are committed by African, Middle Eastern and South East Asian ethnic groups who perform acts of female genital mutilation. Muslims, Christians and atheists inflict the act on girls between the age of 1 day and 16 years within unhygienic conditions usually lacking anaesthetics. It is important also to realise that, despite the claims of Muslim advocates, no religious precedent exists in the Koran<sup>59</sup>.

Three forms of such female genital mutilation exist:

- 1. Circumcision the excision of the clitoral prepuce.
- 2. Excision amputation of the clitoris with or without the labia minora.
- 3. Infibulation removal of the clitoris, labia minora and a large part of the labia majora with closure of the vulva through sewing. Only a small opening remains to allow the expulsion of menses and urine.

The act of surgical modification for purposes other than the treatment of disease was recognised in 1985 and rendered illegal within the United Kingdom under the aforementioned "Prohibition of Female Circumcision Act" 10. However, the





**Fig. 7.17** Examples of breast implant reactions. (a) Fibrosis and reaction to leaked contents. (b) Implant reaction 25 years post-operative, producing a calcified shell enclosing the prosthesis.

increased international movement of individuals around the globe has resulted in more cases being encountered within our multi-cultural society. Such modification unfortunately continues to occur, possibly performed whilst the child is removed from the country under the false pretences of a foreign vacation.

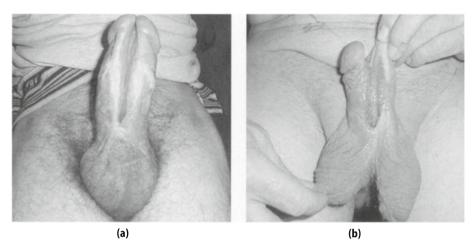
As it stands, the law does not, however, discriminate between such cases of child abuse and self-inflicted alterations performed by consenting adults wishing to alter their own appearance.

Penile modification includes the socially acceptable, though again often unconsented given the age of the individual, circumcision performed for either religious purposes or for reasons of hygiene. Equally, examination of a non-circumcised male body should include the retraction and examination of the foreskin possibly revealing the origin of a metastatic squamous cell carcinoma.

Testicular implants are offered to rebalance asymmetry following orchidectomy, be it for torsion or neoplasia, though often it is not felt necessary by the individual. The prosthesis is ovoid, being usually created from silicone, though newer saline-filled implants have been offered following concerns over the use of the former in implants.

Temporary alterations of the genitalia, especially the scrotum, can be created through the use of injected saline. Similar to play piercing, the act, referred to as "saline injections", is regarded as sexual by the individual and can involve extragenital anatomical sites. The effect resembles that of localised oedema and the fluid is gradually absorbed over subsequent days though the risks of scrotal infections through piercing and poor hygiene are raised. "Vacuum pumping" also temporarily alters the size of genitalia, being the use of a hand-pump device to enhance natural size. The pump device creates a vacuum and the subsequent venous engorgement and oedema increases the dimensions of the anatomical site. The side effects include capillary haemorrhages and the creation of serous fluid containing cutaneous blisters.

The self-inflicted alteration of the male genitalia, including injections and pumping, maintains a degree of popularity as witnessed by numerous dedicated websites. Surgically, the act of subincision remains one of the commonest performed (Fig. 7.18). The procedure involves the surgical opening of the urethral meatus usually along the ventral aspect to produce an artificial hypospadias. This neo-meatal open-



**Fig. 7.18 (a)** An example of subincision of the urethra. (Image courtesy of the individual). **(b)** A combined subincision with bifurcation of the scrotum. (Image courtesy of Split Sack/EnigmaModMan.)

ing may then be extended further down to the base of the penis if the individual so wishes. Aboriginal tribesmen have traditionally performed the act initially during a coming-of-age ceremony<sup>60</sup>. The opening is extended further as the man reaches different milestones in his life. Personal accounts by people possessing such modifications report varied success with respect to erections and ejaculation, though urination is recommended in the seated position or by the passing of a metal tube into the neo-meatus. The reasons for such alterations may be purely aesthetic or part of a fetish that arouses the individual (it is reported that the exposure of the urethral mucosa renders the area more sensitive than previously) or to increase the girth of the phallus. Alternatively, the more extreme modification of the male genitals produces a bifurcation of the entire penis.

## **Gender Reassignment**

Gender reassignment is the most complete form of body modification and is used as a surgical treatment of sexual dysphoria, requiring appropriate documented psychological counselling before and after the procedure. The estimated number of individuals within the United Kingdom who have undergone such transformation has been placed at 5,000 with most having been performed privately, though statefunded operations commonly occur albeit in a few specialist centres. Many individuals travel abroad for the surgery<sup>61</sup>.

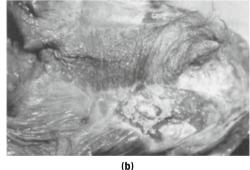
"Male-to-female" alterations are performed through the dissection of the penile skin from the corpora and separation of the glans penis with its associated neurovascular bundle. The urethra is dissected out and a neo-meatus is fashioned below the neo-clitoris created from the glans penis. The testes are removed, with the spermatic cords tied off, and excess erectile tissue excised to prevent subsequent narrowing of the vaginal canal during sexual arousal. The scrotal skin and penile skin is reflected to cover the defect and, if so requested, can be fashioned into a neo-vagina that is passed inside out and secured in the plane between the prostate

and the rectum. If the penile skin is too small to provide an appropriate length of vaginal canal, the option of a colonic mucosa neo-vagina can be offered, requiring additional abdominal surgery (Fig. 7.19). The result is an acceptable appearance with either a v- or w-shaped line of sutures extending from the thigh creases to the anterior pubic region and a more atrophic appearance to the "mucosa" of the introitus and vulva. If created from skin, the neo-vagina may show evidence of hair formation or infected follicles though laser pre-treatment is often performed to minimise this (Fig. 7.19 (b)). Examination of an individual at post-mortem would also reveal the presence of a prostate and seminal vesicles, with tied-off spermatic cords within the inguinal canals.

Additional surgery may be requested including breast augmentation following oestrogen treatment, facial surgery and laryngeal procedures to alter the vocal cord length and to shave the thyroid cartilage, producing a less prominent "Adams' Apple" in profile. It should be noted that similar surgery to fashion a neo-vagina is performed in children with congenital vaginal atresia, though obviously the presence of male internal genitalia would exclude this possibility.

The psychological stresses experienced by transsexuals may result in an increased risk of suicide requiring appropriate toxicological investigations. Iatrogenic causes of sudden death should also be considered, notably the increased rate of pulmonary thromboemboli secondary to the use of exogenous oestrogen preparations by these individuals, although the increased availability of oestrogen dermal patches





**Fig. 7.19 (a)** Intra-operative image of gender reassignment. The neo-clitoris and refashioned urethra are evident. The individual photographed declined the option of a neo-vagina. (Photograph courtesy of Mr. T. Terry, Leicester General Hospital, UK.) **(b)** A neo-vagina opened at post-mortem examination showing hair follicles along its entire length, with areas of ulceration.

has reduced this rate in recent years<sup>62,63</sup>. Oestrogen therapy can also result in hyperprolactinaemia, accounting for an appreciably enlarged pituitary gland upon radiological or internal examination<sup>64</sup>.

"Female-to-male" alterations are created through the use of hormones, implants and surgery. Exogenous testosterone can create a markedly hypertrophic clitoris that may be dissected and fashioned into a glans penis. Metoidioplasty aims to create a functioning urethra enclosed within the neo-glans but are generally too small for the purpose of implants. Phalloplasties require skin grafts usually from the non-dominant forearm but other options include the lower limb or pedicles from the lower abdominal wall. A neo-urethra is formed around a Foley catheter and sutured into place. The labia are sutured together to produce a scrotum. The internal genitalia are removed as a total abdominal hysterectomy with bilateral salpingo-oophorectomy, although some centres offer the surgery laparoscopically. The procedures may be staggered or performed at a single operation, with implants added into the scrotum and devices placed in the neo-phallus similar to those used in the treatment of impotence such as rigid rods or "pumps". Breasts are removed to create a male-type appearance. Despite a theoretical increased risk of ischaemic heart disease relating to the use of testosterone, few long-term side effects have been documented<sup>63</sup>.

## **Decorative Implants**

Decorative subcutaneous implants are increasingly popular due to modern-day folklore. The insertion of materials under the skin to produce a three-dimensional pattern has been revived and even furthered, though the history can be traced to many tribes where stones or seeds were passed under the skin to produce designs. Eighteenth-century Japanese gamblers, later known as the Yakuza, developed the art with "beading" or "pearling"; the insertion of beads under the penile skin. This act has become popularised amongst Russian soldiers and prisoners, and in the West due in part to "gangster-chic" with contemporary beads composed of Teflon or inert metal<sup>65,66</sup>. The Yakuza were also infamous for tattoos, one for each crime committed, and the self-induced amputation of digits to demonstrate allegiance to their clan.

Transdermal implants may not be confined purely to the genitals or be entirely decorative in themselves, such as those constituting fixation points set over bony prominences to allow the insertion of separate decorative pieces such as metal spikes or false horns placed into the scalp.

## **Apotemnophilia**

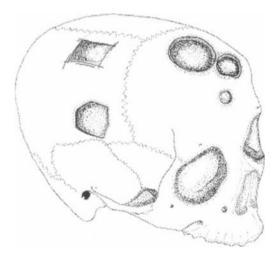
Apotemnophilia is described as a paraphilia in which an individual experiences a fetish for amputees and may result in that person wishing to have part of their anatomy removed. As such, self-amputation may be the most extreme form of body modification. The acts range from the aforementioned removal of digits or self circumcision to the deliberate removal of entire limbs, castration or penectomy, though it should be pointed out that the latter does not usually equate with a desire to be transsexual. The psychology behind these fetishes has been infrequently published and as such is poorly understood, <sup>67</sup> possibly representing a form of Body Dysmorphic Disorder (BDD). Despite the little documentation in the literature the fetish is not uncommon, as reflected by the prevalence of pornography and web-

sites devoted to it<sup>67</sup>. In response to the recognition of this psychiatric illness, surgical amputation of normal limbs may be performed under clinical situations, albeit controversially<sup>67-69</sup>, although sheer desperation on the part of the sufferer may result in self-induced attempts often with poor results. Differentiation from torture, psychotic mental illness, suicide attempts or amputation for a physical reason is essential and a positive documented history of paraphilia may assist in these matters<sup>70,71</sup>.

## **Trephination**

Trepanning, trephining or trephination refers to the act, either therapeutic or otherwise, of boring or sawing an aperture into bone, though it usually refers to the calvarium, through the use of a specialised instrument referred to as either a trephine or a trepan. Examples of such modifications have been described in Neolithic and ancient South American burials, many with evidence of healing indicating survival of the individual and to a degree is still practiced in East Africa<sup>72–75</sup> (Fig. 7.20).

The act continues to have a modern Western following since its apparent revival in the mid-twentieth century. Advocates believe that when the cranial sutures fuse in childhood the intracranial pressure rises, preventing the natural pulsation of the brain. By creating an opening in the skull this pressure is relieved, thus allowing an increased cerebral blood flow to produce an altered level of consciousness. The surgery is performed either in the home, through the use of electrical tools, or possibly within a clinical setting. The operation requires the removal of a bone fragment without damage to the underlying meninges or brain parenchyma, therefore aiming to limit the risk of meningitis or abscess formation. Despite an on-line International Advocacy Group the true incidence of such modifications and their associated complications remains unknown<sup>76</sup>.



**Fig. 7.20** A diagram illustrating the variations on trephination: a) Ancient methods, using stone or metal tools to remove part of the calvarium over the parietal, temporal and frontal areas, and b) Modern site, being central in the frontal area, usually through the use of electric tools.

# **Summary**

A variety of forms of body art and body modification are currently being practiced throughout the world. Often, these acts possess ritual or traditional origins dating back millennia. Despite this, connections to specific ethnic groups may be inferred by the presence of such decorations although the practices have become so widespread as to make such conclusions relatively inaccurate.

It is also important not to view the art forms with any prejudice or judge the social life of the deceased based on these; the oft-cited link between tattoos and violent death or criminal behaviour remains generally unproven. Recent surveys revealed that more women are being tattooed, especially middle-class professionals. Many also possess body piercings other than ear-rings, notably umbilical decorations. The changes in fashions have brought these once "underground" acts into mainstream life and all social classes (as proven by the lingual piercing displayed by a younger member of the British Royal Family). Also, the risks of blood-borne viral diseases, though potential, are less in those with professional compared to non-professional tattoos and an increased incidence may be in part due to other practices, notably the abuse of intravenous narcotics.

# **Acknowledgements**

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## References

- 1. Tait, H. (Ed) Seven thousand years of jewellery. British Museum Publications, 1989.
- 2. Makkai T, McAllister I. Prevalence of tattooing and body piercing in the Australian community. Commun Dis Intell 2001;25(2):67–72.
- 3. Ferguson H. Body piercing. BMJ 1999;319:1627-9.
- 4. Body Modification Ezine. http://www.bmezine.com
- 5. De Moor RJG, De Witte AMJC, De Bruyne MAA. Tongue piercing and associated oral and dental complications. Endod Dent Traumatol 2000;16:232–7.
- Botchway C, Kuc I. Tongue piercing and associated tooth fracture. J Ir Dent Assoc 2001;47(1):10-13.
- Willmott FE. Body piercing: lifestyle indicator or fashion accessory? Int J STD AIDS 2001;12(6):358-60.
- 8. Miller L, Edenholm M. Genital piercing to enhance sexual satisfaction. Obstet Gynecol 1999;93(5):837.
- 9. Koenig LM, Carnes M. Body piercing medical concerns with cutting-edge fashion. J Gen Intern Med 1999;14(6):379–85.
- Her Majesty's Stationery Office. The Prohibition of Female Circumcision Act, 1985. HMSO, London, 1985.
- 11. World Health Organisation. Female Genital Mutilation Report of a WHO Technical Working Group, Geneva, 1995.

- 12. Ramage IJ, Wilson N, Thomson RB. Fashion victim: infective endocarditis after nasal piercing. Arch Dis Child 1997;77(2):187.
- 13. Oschenfahrt C, Fiedl R, Hannekum A, Schumacher BA. Endocarditis after nipple piercing in a patient with a bicuspid aortic valve. Ann Thorac Surg 2001;71(4):1365-6.
- Khanna R, Kumar SS, Raju BS, Kumar A. Body piercing in the accident and emergency department. J Accid Emerg Med 1999;16(6):418-21.
- 15. Pugatch D, Mileno M, Rich JD. Possible transmission of human immunodeficiency virus type 1 from body piercing. Clin Infect Dis 1998;26(3):767–8.
- Hadfield-Law L. Body piercing: issues for A & E nurses. Accident and Emergency Nursing 2001;9:14-19.
- 17. Rutty J. Essential of autopsy practice, vol 1, Rutty GN, editor. London: Springer Verlag, 2002.
- 18. Chung WH, Wang CM, Hong HS. Allergic contact dermatitis to temporary tattoos with positive para-phenylenediamine reactions: report of four cases. Int J Dermatol 2001;40(12):754-6.
- 19. Raupp P, Hassan JA, Varughese M, Kristiansson B. Henna causes life-threatening haemolysis in glucose-6-phosphate dehydrogenase deficiency. Arch Dis Child 2001;85(5):411–2.
- 20. Pumphrey RS, Roberts IS. Post-mortem findings after fatal anaphylactic reactions. J Clin Pathol 2000;53(4):273-6.
- 21. Price N, Gottfried MR, Clary E, Lawson DC, Baillie J, Mergener K, Westcott C, Eubanks S, Pappas TN. Safety and efficacy of India ink and indocyanine green as colonic tattooing agents. Gastrointest Endosc 2000;51(4 Pt 1):438-42.
- 22. Knight B. The coroner's autopsy. Churchill Livingstone Publishers, 1983.
- 23. Whittaker DK, MacDonald DG. A colour atlas of forensic dentistry. London: Wolfe Publishing, 1989.
- 24. Tschirhart DL, Noguchi TT, Klatt EC. A simple histochemical technique for the identification of gunshot residue. J Forensic Sci 1991;36(2):543–7.
- 25. Masser MR, Di Meo L, Hobby JA. Tattooing in reconstruction of the nipple and areola: a new method. Plast Reconstr Surg 1989;84:677–81.
- Blancho-Davila RS. Bianchi tattoo in Ancient Egypt. In: Marks of civilisation: Artistic transformation of the human body. Los Angeles: University of California. 1985;21–28.
- 27. Guarino C, Casoria P, Menale B. Cultivation and use of Isatis tinctoria L. (Brassicacae) in Southern Italy. Economic Botany 2000;54(3):395–400.
- 28. Simmons DR. Ta Moko: The art of Maori tattoo. Auckland: Reed Methuen, 1986.
- 29. Millner VS, Eichold BH. Body piercing and tattooing perspectives. Clin Nurs Res 2001; 10(4):424-41.
- 30. Silverman AL, Sekhon JS, Saginaw SJ, Wiedbrauk D, Balasubramaniam M, Gordon SC. Tattoo application is not associated with an increased risk for chronic viral hepatitis. Am J Gastroenterol 2000;95:1312–15.
- 31. Nishioka S de A, Gyorkos TW, Joseph L, Collet JP, Maclean JD. Tattooing and risk for transfusion-transmitted diseases: the role of the type, number and design of the tattoos, and the conditions in which they were performed. Epidemiol Infect 2001 Feb;128(1):63–71.
- 32. Horney DA, Gaither JM, Lauer R, Norins AL, Mathur PN. Cutaneous inoculation tuberculosis secondary to "jailhouse tattooing". Arch Dermatol 1985;121(5):648-50.
- 33. Sperry K. Tattoos and tattooing. Part II: Gross pathology, histopathology, medical complications, and applications. Am J Forensic Med Pathol 1992;13(1):7–17.
- 34. Burton J, Rutty GN. The hospital autopsy, 2nd ed. London: Arnold, 2001.
- 35. Kuperman-Beade M, Levine VJ, Ashinoff R. Laser removal of tattoos. Am J Clin Dermatol 2001;2(1):21-5.
- 36. Angres GG. Eyeliner implants: A new cosmetic procedure. Plast Reconstr Surg 1984;73:833-6.
- McKee PH. Pathology of the skin with clinical correlations, 2nd ed. London: Mosby-Wolfe, 1996.
- 38. Anderson LL, Cardone JS, McCollough ML, Grabski WJ. Tattoo pigment mimicking metastatic malignant melanoma. Dermatol Surg 1996 Jan;22(1):92–4.
- 39. Maury FF, Dulles CW. Tattooing as a means of communicating syphilis. Am J Med Sci 1878;75:44.
- 40. Jones MS, Maloney ME, Helm KF. Systemic sarcoidosis presenting in the black dye of a tattoo. Cutis 1997;59(3):113–5.
- 41. Sowden JM, Cartwright PH, Smith AG, Hiley C, Slater DN. Sarcoidosis presenting with a granulomatous reaction confined to red tattoos. Clin Exp Dermatol 1992;17(6):4468.
- 42. Wiener DA, Scher RK. Basal cell carcinoma arising in a tattoo. Cutis 1987;39(2):125-6.
- 43. Soroush V, Gurevitch AW, Peng SK. Malignant melanoma in a tattoo: case report and review of the literature. Cutis 1997 Mar;59(3):111–12.
- 44. Petit F, Divaris M, Guilbert F. Unusual breast scars. Ann Chir Plast Esthet 1999;44(6):649-50.

- 45. Alam M, Omura NE, Dover JS, Arndt KA. Glycolic acid peels compared to microdermabrasion: a right-left-controlled trial of efficacy and patient satisfaction. Dermatol Surg 2002;28(6):475–9.
- 46. Coleman III WP. Dermal peels. Dermatol Clin 2001;19(3):405-11.
- 47. Freeman MS. Microdermabrasion. Facial Plast Surg Clin North Am 2001;9(2):257-66.
- Lent WM, David LM. Laser resurfacing: a safe and predictable method of skin resurfacing. J Cutan Laser Ther 1999;1(2):87–94.
- 49. Sposito MM. New indications for botulinum toxin type a in cosmetics: mouth and neck. Plast Reconstr Surg 2002;110(2):601–11.
- 50. Gilliland MD, Coates N. Tumescent liposuction complicated by pulmonary oedema. Plast Reconstr Surg 1997;99(1):215–9.
- 51. Gibbons MD, Lim RB, Carter PL. Necrotizing fasciitis after tumescent liposuction. Am Surg 1998;64:458-60.
- 52. Talmor M, Barie PS. Deaths related to liposuction. N Engl J Med 1999;341(13):1001.
- 53. Talmor M, Hoffman LA, Lieberman M. Intestinal perforation after suction lipoplasty: a case report and review of the literature. Ann Plast Surg 1997;38:169–72.
- 54. Moran M. Lidocaine dose questioned in liposuction deaths. American Medical News 1999;30:32.
- 55. Laub DR Jr, Laub DR. Fat embolism syndrome after liposuction: a case report and review of the literature. Ann Plast Surg 1990;25(1):48–52.
- 56. Platt MS, Kohler LJ, Ruiz R, Cohle SD, Ravichandran P. Deaths associated with liposuction: case reports and review of the literature. J Forensic Sci 2002;47(1):205–7.
- 57. Rao RB, Ely SF, Hoffman RS. Deaths related to liposuction. N Engl J Med 1999;340(19):1471-5.
- 58. Klein JA. Deaths related to liposuction. N Engl J Med 1999;341(13):1001.
- Andrews G. (Ed) Women's sexual health, 2nd ed. London: Bailliere Tindall (Harcourt), London, 2001.
- Pounder DJ. Ritual mutilation: Subincision of the penis among Australian Aborigines. Am J Forensic Med Pathol 1983;4(3):227-9.
- 61. Terry T. Personal Communication. 2002.
- 62. Asscheman H, Gooren LJ, Eklund PL. Mortality and morbidity in transsexual patients with cross-gender hormone treatment. Metabolism 1989;38(9):869-73.
- 63. van Kesteren PJ, Asscheman H, Megens JA, Gooren LJ. Mortality and morbidity in transsexual subjects treated with cross-sex hormones. Clin Endocrinol 1997;47(3):337-42
- 64. Asscheman H, Gooren LJ, Assies J, Smits JP, de Slegte R. Prolactin levels and pituitary enlargement in hormone-treated male-to-female transsexuals. Clin Endocrinol 1988;28(6):583-8.
- 65. Djajakusumah TS, Meheus A. Artificial nodules of the penis: case report of an Indonesian man. Sex Transm Dis 2000;27(3):152-3.
- 66. Rothschild MA, Ehrlich E, Klevno WA, Schneider V. Self-implanted subcutaneous penile balls a new phenomenon in Western Europe. Int J Legal Med 1997;110(2):88–9.
- 67. Wise TN, Kalyanam RC. Amputee fetishism and genital mutilation: case report and literature review. J Sex Marital Ther 2000;26(4):339-44.
- 68. Dyer C. Surgeon-amputated healthy legs. BMJ 2000;320:332.
- Fisher K, Smith R. More work is needed to explain why patients ask for amputation of healthy limbs. BMJ 2000;320:1147.
- 70. Hall DC, Lawson LG, Wilson LG. Command hallucinations and self-amputation of the penis and hand during a first psychotic break. J Clin Psychiatry 1981;42(8):322-4.
- 71. Madea B. Homizidale versus suizidale Penisamputation. Ein Beitrag zum Problem der fehlenden und irrefuhrenden Spuren. [Homicidal versus suicidal penis amputation. A contribution to the problem of missing and conflicting stains.] Archiv Fur Kriminologie 1995;196(3-4):70–76.
- 72. Furnas DW, Sheikh MA, van den Hombergh P, Froeling F, Nunda IM. Traditional craniotomies of the Kisii tribe of Kenya. Ann Plast Surg 1985;15(6):538-56.
- 73. Janssens PA. Paleopathology: Diseases and injuries of prehistoric man. London: John Baker, 1970.
- 74. Stone J, Miles M. Skull trepanation among the early Indians of Canada and the United States. Neurosurgery 1990;26:1015–20.
- Marino R Jr, Gonzales-Portillo M. Preconquest Peruvian neurosurgeons: a study of Inca and pre-Columbian trephination and the art of medicine in ancient Peru. Neurosurgery 2000; 47(4):940-50.
- 76. The International Trepanation Advocacy Group http://www.trepan.com

A.M. Davison

## Introduction

A wound is defined as an injury to the surface of the body, "caused by a cut, blow, hard or sharp impact etc., especially one in which the skin is cut or broken; an external injury." In practice, to the medical practitioner, the terms wound and injury are synonymous but strictly the legal definition of a wound involves breaking of the full thickness of the skin. An incision is a division made by cutting and a cut is defined as a long, narrow opening in a surface, especially the skin, made by something sharp; an incision, a gash. The verb to cut means to penetrate or wound with a sharp-edged implement and therefore cuts/incisions are sharp force injuries. To stab is to wound or kill a person by a thrust with the point of a knife (or other weapon) and although it is accepted that fatal stabbing can result from penetration of the body by a blunt weapon, the majority of such cases, certainly in the UK, are caused by sharp-edged weapons, principally knives. Therefore, most (but not all) stab wounds are a form of incised wound.

Incised (cut and stab) wounds can be caused accidentally or deliberately; the latter may be self-inflicted or caused by another party. Of all sharp force trauma fatalities approximately 80% are classified as homicide, 17-19% as suicide and only about 2-3% as accident<sup>2-4</sup>. With regard to homicide, the scale of the problem, and thus the importance to the practice of forensic autopsy pathology, is indicated by the criminal statistics. In England and Wales, homicide by use of a sharp instrument remains the most common method, accounting for 219 of 761 victims (30% of all homicides; 33% of males, 24% of females) in the year 1999/2000. Although the actual number of cases is similar to that in 1992 (218) the percentage of homicides due to sharp force trauma has fallen from 38%<sup>5</sup>. The figures for Scotland are even worse; not only is the homicide rate almost twice that of England and Wales<sup>6,7</sup>, but in the period 1989-98, 44% were the result of sharp force injury. Similarly, the homicide rate in Glasgow is nearly twice that in London<sup>8</sup>, a difference which may be largely explained by the persistence of a "knife culture", particularly among young males in Strathclyde - where two thirds of the homicides in Scotland occur. The Scottish Trauma Audit Group (STAG) assesses the care of virtually all trauma patients in Scotland who are hospitalised for at least three days or die as a result of their injuries, although the study excludes subjects pronounced dead at the scene or within 15 minutes of arriving in hospital. Penetrating injuries account for 5.7% of all trauma and 47% of assaults. In Scotland, the vast majority of penetrating trauma is due to sharp force; 76% are the result of an assault and the absolute numbers have been steadily increasing since 1993 (STAG, 2001, personal communication). Various initiatives have been tried to combat the rising number of sharp force assaults but these have met with limited and temporary success<sup>9</sup>. In Sweden, a law restricting the carrying of knives in certain places did not significantly influence the number of sharp force homicides<sup>10</sup>. It should be stressed, however, that the homicide figures would be much worse were it not for improved survival as a result of prompt and skilled medical intervention. Victims who in the past would have died of their injuries now survive and some have been successfully treated for stab wounds to the heart.

Although a significant number of sharp force trauma fatalities are due to self-inflicted wounds, this method is relatively uncommon, being employed in less than 4% of suicides. Cuts are more frequent than stab wounds; there is a male predominance (3:1) and many victims (up to 60%) have a history of mental disorder<sup>2,3,11,12</sup>. Arms are the commonest site of injury; fewer subjects choose the neck or chest/abdomen, and although in most cases wounds are restricted to a single site, the majority (69%) show more than one wound<sup>13</sup>.

This chapter discusses the pathological interpretation of cuts and incised stab wounds. Many readers will encounter these only at autopsy but some may be asked to provide an opinion on non-fatal injuries, either by personal examination of a live subject or review of photographs and documents. In fatalities due to incised wounds the cause of death often presents less of a problem than the other issues that may be raised; force required, manner of infliction, type of weapon, possibility of survival, capacity for movement.

#### The Cut

Known colloquially as a "slash", the meaning of which seems readily understood by lay people, cuts are often incorrectly described by medical practitioners as "lacerations". It is important to distinguish these two types of injury as accurate classification can provide clues to the nature of an assault and possibly also the weapon. A laceration is a tear and implies the application of blunt force; either the victim hit something blunt or vice versa. When the term is used to describe a skin wound, the edges are usually irregular and abraded (grazed), bruising is often present and soft tissue structures (blood vessels, nerves) in the depths of the wound appear crushed. In contrast, the edges of a cut show clean division of the skin and underlying soft tissues (Fig 8.1) but no associated abrasion, bruising or crushing, although "chopping" injuries - inflicted by a heavy weapon with a relatively blunt blade - may exhibit the appearances of both a laceration and a cut. However, while most wounds can be identified as one or the other by naked-eye inspection, occasionally, misinterpretation occurs<sup>14</sup> and magnification, using either a hand lens or dissecting microscope, may be useful. While it could be argued that cases which come to a civil or criminal court should be assessed by experts in wound interpretation, reliance is often placed on the description of a wound by a relatively inexperienced doctor; good quality photographs are not usually available. The categorisation of a wound as either a cut or laceration may be consistent or inconsistent with the alleged altercation, e.g., was the victim assaulted by somebody wielding a hammer - producing a laceration, or a knife - producing a cut?

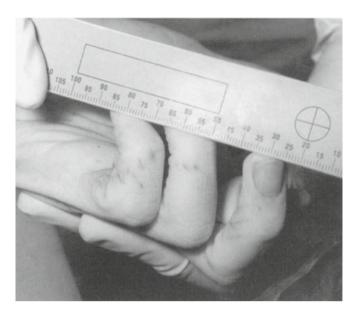


**Fig. 8.1** A "slash" on the left cheek with a tail abrasion at the end nearest the mouth and a "nick" on the upper border. Death occurred due to a stab wound to the neck.

The outcome of a trial and length of custodial sentence may depend on such matters<sup>15</sup>.

Cuts are conventionally differentiated from incised stab wounds by comparing the length of the surface injury with the depth of the wound, i.e., the degree of penetration into the body. In cuts, the length is greater than the depth; the opposite applies to stab wounds. Cuts are not usually fatal unless the neck or wrists are involved and the surface characteristics are not often specific for a particular implement, although if a serrated blade is drawn across the skin at an angle – rather than perpendicular – to the surface, parallel linear abrasions may be seen (Fig 8.2). In some cases the pattern may suggest the use of a "survival" knife<sup>16</sup>. However, usually all that can be said is that the injury was caused by a sharp edge, probably a knife but possibly glass or broken china.

Glass injuries tend to be irregular and ragged and although the wounds are usually of varying depths, body cavities are rarely penetrated. In one study of Accident and Emergency (A & E) admissions due to injuries caused by assault, broken drinking glasses were more commonly the weapons of use than knives<sup>17</sup>. It would seem that the aim of such assaults is to maim rather than kill; the face is commonly involved (72% of cases) and hospitalisation is less often required than after a knife assault<sup>17,18</sup>. Such studies provided a catalyst for the increasing use of tempered (toughened) glass and/or plastic glasses in an attempt to decrease the number of such injuries. A & E medical personnel and pathologists should remember that some types of glass are radio-opaque and therefore retained fragments may be detected on x-ray.



**Fig. 8.2** Parallel linear abrasions and superficial cuts on the back of three fingers caused by the serrated blade seen in Fig 8.10b.

The vast majority of deaths due to cuts are cases of suicide<sup>3</sup>, although determining the manner of infliction, i.e., how the injuries occurred, may not be possible from the pathology evidence alone. The location, number and pattern of incised wounds along with evidence of other injuries, e.g., a fractured larynx from pressure on the neck, may provide substantial clues but in many cases the pathologist would be well advised to interpret the findings with caution until the full circumstances are known. In particular, the medical history of the victim, the clothing and the "scene" may be relevant. Strictly, the manner is determined by the outcome of the legal process – inquest, inquiry or criminal trial – but great reliance is often placed on the pathologist's opinion.

Self-inflicted cuts are not usually fatal unless the neck is involved although death can certainly occur from "slashed" wrists, particularly if medical attention is not sought. Where the victim has falsely claimed to have been assaulted, cuts are typically superficial, clustered in groups and absent from the nose, ears, eyes and lips. Superficial parallel cuts, or old scars, across the front of the wrist are consistent with a suicidal gesture and probably indicate a personality disorder; there may be a history of previous wounding, overdose, depression or recent argument with a partner. Alternatively, the perpetrator of an assault may have inflicted the injury (on his/her body) to bolster a claim of self-defence<sup>10</sup>. Where there is a determination to commit suicide success is often obtained by another method, e.g., hanging or drug overdose, and it is not uncommon in such cases to see superficial parallel cuts across the front of the forearm/wrist (Fig 8.3); presumably a first attempt before realising that it was either too painful or not likely to be successful. At autopsy, dissection of the soft tissues should be performed to assess the damage to underlying structures such as blood vessels, nerves and tendons, which may help to address questions regarding blood loss and capacity to hold a weapon.

Fatalities due to self-inflicted cuts to the neck do occur but the pathological appearances may simulate a homicide. Typically, in a suicide, the cuts will be parallel and tentative, "hesitation" superficial incised wounds will be present; they have been described in 62–80% of such cases<sup>2,11,19</sup>. However, a deep cut – even down to the spine – and an absence of "hesitation" wounds does not preclude a suicide, although more than one deep wound and the presence of "defence" injuries (see below) should raise suspicion. In such cases, "peripheral" evidence may be more helpful with regard to interpretation of the injuries: presence and location of a knife at the scene, suicide note, security of the scene, toxicology results, medical history. Self-inflicted slashing of the abdomen (harakiri) is quite rare – even in Japan – and is not a terribly effective method of suicide<sup>12,20,21</sup>.

Cuts inflicted by another party do not often form a pattern; they may appear haphazard – often crossing each other – and are unlikely to be parallel (Fig 8.4). They usually deepen rapidly, and classically there is no "hesitant" quality, although occasional cases of homicide exhibiting marks interpreted as "tentative" have been described<sup>22,23</sup>, with several suggested explanations for such a finding: physical disparity between victim and assailant, intoxication of the assailant, use of a blunt knife, infliction after the victim has been rendered unconscious - perhaps as a ritual marking of the body. The direction of the slash can sometimes be inferred from the presence of a "tail" abrasion; a superficial linear graze caused by the tip or cutting edge not fully penetrating the skin, usually as the knife comes away from the body rather than at the first point of contact (Fig 8.1). Occasionally, two or more cuts may appear aligned but separated by intact skin; the pathologist should be aware that several wounds may be caused by one "slashing" action (Figs 8.5 (a) & (b)). This can be important in reconstructing events and may be a consequence of several factors: movement of the deceased, local anatomy e.g. curved surface, bony ridges, and/or nature of the weapon e.g. shape and bluntness of the blade.



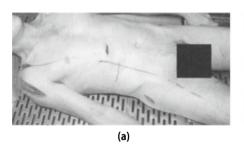
**Fig. 8.3** Recent self-inflicted, parallel, superficial, incised wounds on the front of the forearm in a case of hanging.

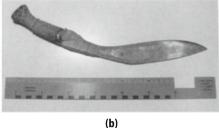


**Fig. 8.4** Homicidal "cut-throat" wounds of varying depths, showing a haphazard appearance. The trachea (T) was severed and the oesophagus, strap muscles, right common carotid artery and right internal jugular vein were injured.

The direction and depth of neck wounds rarely help to distinguish suicide from homicide, and it is usually not possible to categorically state which hand held the knife and whether homicidal wounds were inflicted from in front or behind. For instance, a homicidal cut inflicted from behind with the knife in the right hand may be the same as one inflicted from the front with the knife in the left hand. Therefore, over-interpretation should be avoided; it is often better to wait until mechanisms of infliction are suggested before commenting on these matters. What can be said about homicidal neck wounds is that when inflicted from behind, they usually start higher on the opposite side to the hand which holds the knife, moving down across the neck to the other side<sup>24</sup>, although as this also probably applies to many self-inflicted wounds<sup>25</sup> this information may be of limited value.

Cuts may qualify as "defence-type" wounds if they occur on a part of the body which could have been used by the victim in an attempt to protect another (vital or more important) part of the body, e.g., the head, or in an attempt to grasp the weapon from an assailant. They occur in 38.5–47% of sharp force homicides, seem to be more common in female victims and are rarely seen in deaths due to a single stab wound 19,26–29. Common sites include the ulnar borders of the forearms (Fig 8.6 (a)) and both surfaces of the hands (Figs 8.2, 8.6 (b)); less commonly, they occur on the outer aspects of the upper arms and legs. Injuries on the back of the forearm or hand have been described as "passive" and are more common than those on the palm or between the fingers, which have been described as "active" Due to the often haphazard nature of an assault the pathologist should indicate that wounds





**Fig. 8.5** (a) Incised wounds of the right shoulder, trunk and thigh. Note the discontinuous nature of the wound which started over the outer right clavicle and extended as a superficial cut down the chest and abdomen onto the front of the thigh. Two stab wounds were also present; in the epigastrium and over the right hip. (b) The Ghurka knife (kukri) which caused the injuries in Fig. 8.5a and 8.13. The knife weighed 360 g, the tip and single cutting edge were moderately sharp.

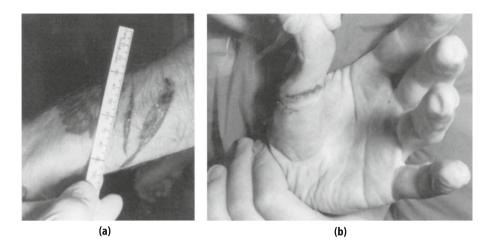
in these sites are consistent with – rather than diagnostic of – attempts at defence by the victim. Furthermore, cuts to the palm can also be seen in suicide<sup>2,12</sup>. The relevance of genuine "defence-type" injuries is that they suggest the victim was conscious at some time during the assault and was probably not caught completely by surprise. Conversely, Spitz<sup>30</sup> suggests that the absence of "defence-type" wounds raises the possibility that the victim was restrained.

Accidental cuts are common and usually minor. Fatalities can occur when falls occur through glass windows<sup>4</sup>, perhaps severing a major limb artery, or in a work environment when knives slip or the sharpness of a blade is under-estimated.

## The Incised Stab Wound

In the majority of autopsies where death is primarily due to a stab wound, the manner is homicide; suicide and accidents<sup>31,32</sup> do occur but both are relatively rare. The circumstances usually allow accidents to be recognised as such but differentiating homicide from suicide can occasionally be problematic.

Stab wound victims – and their assailants – are usually young, adult males 10,28,33,34. The most popular weapon is a kitchen or sheath knife, although as with cuts, anything with a sharp edge, e.g., glass, can produce an incised stab wound<sup>32,35,36</sup>. Stab wounds are generally more dangerous than cuts because they are more likely to penetrate body cavities, injuring internal organs and associated blood vessels. The surface wound may be small and there may not be copious external haemorrhage or physical signs of internal damage, which might partly explain how the severity can be underestimated by lay witnesses and medical personnel<sup>37,38</sup>. A recent case report<sup>39</sup> described how a stab wound to the temple was initially misdiagnosed as a "slash" only for a retained fragment of the blade to be detected on a skull x-ray performed during the second visit to hospital. However, the fatality rate (deaths per number of stabbing incidents) is relatively low, about 17%<sup>33</sup>, because although stabbing is the commonest cause of homicide in the UK, there are many knife-related assaults, and survival has improved, largely due to better trauma care. Indeed, the fact that more victims survive - at least long enough to be admitted to hospital poses problems to the pathologist that were less frequently encountered in the past, e.g., identification of marks of medical intervention and interpretation of treatment.



**Fig. 8.6 (a)** Two incised "defence-type" wounds on the outer lower forearm. **(b)** An incised "defence-type" wound at the base of the thumb, consistent with the victim trying to grab the knife blade from the assailant.

A pathologist may subsequently be asked if the wounds were "necessarily fatal" and if not, did the medical treatment (or lack of treatment) contribute to the death? The autopsy interpretation of stab wounds usually requires the pathologist to collate information from various sources before producing a comment designed to answer (as best as possible) questions that might be raised in court. In addition to the accurate examination and recording of external and internal injuries, assessment of the suspect weapon (if available), clothing and scene may provide vital clues.

## **Number of Wounds**

The number of stab wounds may assist in determining the manner of death. One would not usually expect there to be more than one in an accident but differentiating homicide from suicide may be difficult as both may occur as the result of single or multiple stab wounds. Indeed, homicides involving a single stab wound occur not infrequently – 36–55% of all homicidal stabbings<sup>27,29,34,40,41,42</sup> – and in such cases the defence legal team usually suggest that the accused did not intend to kill the victim. Interestingly, it has been observed that a single external wound may be associated with more than one wound in the anterior wall of the heart<sup>43</sup>. This may be the result of a "double thrust" by the assailant without complete removal of the knife, but bearing in mind the dynamic nature of any altercation and in particular, movement of the victim, it would be unwise to conclude that there had definitely been two separate stabbing actions by the assailant. In addition, similar findings – multiple internal tracks associated with one external wound – have also been described in suicide<sup>3,44</sup>.

It has been reported that the majority (64%) of suicidal stabbings are the result of a single wound and it therefore follows that a significant minority are associated

with multiple stab wounds<sup>3</sup>. Indeed, some cases may exhibit a surprisingly large number; Karlsson et al.<sup>11</sup> described a maximum of 31 and Start et al.<sup>3</sup> reported one case with 85 stab wounds. Multiple stab wounds do not therefore necessarily indicate a homicide, although the finding of more than one "fatal" wound should make the pathologist cautious.

A large number of potentially fatal wounds, possibly with other types of trauma, e.g., blunt injuries, may suggest a homosexual element/motivation<sup>45,46</sup> and makes a successful defence of accidental infliction less likely. Although the "F" word (frenzied) should be avoided<sup>47</sup> and pathologists are recommended not to comment on intent, most jurors will draw conclusions (as to intent) partly based on the number of wounds. It has been claimed that more than 10 stab wounds suggests a close relationship between victim and assailant or mental illness in the perpetrator<sup>10</sup>.

# **External Wound Characteristics: Site, Shape and Size**

#### Site

The anatomical location of a stab wound is one factor which determines the likelihood of a fatal outcome. When considering assaults, the chest is a popular target and bearing in mind the internal structures, e.g., heart, lungs and great vessels, it seems surprising that any victims survive. The abdomen is also a reasonably popular site but the head and neck are less often involved, although individually they are probably more dangerous<sup>27,28,29,33,34,40</sup>. Stab wounds to the limbs are less frequently fatal and usually only when the femoral arteries are involved. In Australia, "unexpected" fatalities have been described following stabbing of the thigh as a traditional form of punishment in the Aboriginal community<sup>48</sup>. Stab wounds to the back and flank are also considered to be of relatively low risk to life<sup>49</sup> but wounds to the buttocks may be misdiagnosed or not fully investigated and deaths can occur due to visceral or vascular injury<sup>50</sup>. Injuries that occur in the upper gluteal region (above a line through the greater trochanters) are associated with more internal damage than those in the lower zone<sup>51</sup>.

Most suicidal stab wounds are to the front of the trunk, particularly the left chest<sup>2</sup>, while the head, neck and extremities are rarely involved<sup>3,44,52</sup>.

At autopsy, the position of each stab wound has to be described so that it can be readily understood by fellow medical experts, lawyers and – most importantly – jurors. It is customary to provide measurements from a named part of each wound (usually one end) to adjacent fixed anatomical landmarks, e.g., point of shoulder, edge of ribcage, and to the midline of the body and the heel, although the latter is of dubious significance as it is extremely unlikely to be of help in identifying the assailant or excluding a suspect.

## **Shape**

Wound shape and size depend on several factors: type of weapon, direction of thrust, movement of blade and victim, tension of skin, and orientation of elastic, collagen and muscle fibres. The latter determine the so-called cleavage lines of the skin (Fig 8.7)<sup>53</sup>.

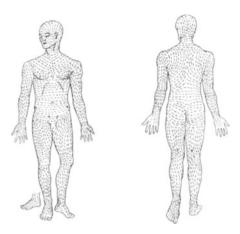
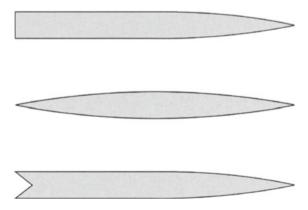
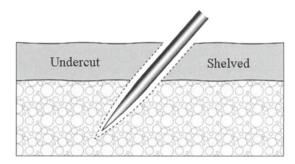


Fig. 8.7 Cleavage lines of the skin as described by H.T. Cox<sup>53</sup>. Reproduced courtesy of Blackwell Science Ltd.

The wounds need to be described in detail, paying particular attention to: ends, overall shape, edges (or margins) and damage (bruising/abrasion) to the surrounding skin. The ends may be sharp or blunt/squared. Many wounds are simple slits, either with two sharp ends or one blunt and one sharp end. Most knives have a single sharp cutting edge and a broader non-cutting edge, the latter is responsible for the squared end of a wound or a "fishtail" appearance due to splitting of the skin, although it may also produce a sharp end – particularly in superficial stab wounds – as many single-edged blades have a short segment at the tip where both edges are sharp (Fig 8.8). The true shapes of the ends are best determined when the margins are apposed but they may be difficult to assess; both may appear rounded, particularly if drying has occurred, the knife has a blunt cutting edge or the blade enters the body up to the ricasso – the short blunt section of the blade between the cutting edge and the handle of the knife<sup>25</sup>. Wounds produced by the same knife may be irregular, sometimes "L" or "V" shaped, suggesting an angled blade at



**Fig. 8.8** Common appearances of stab wounds caused by a single-edged blade.



**Fig. 8.9** Undercutting and shelving of skin and soft tissue at stab wound edges due to angled entry of the weapon.

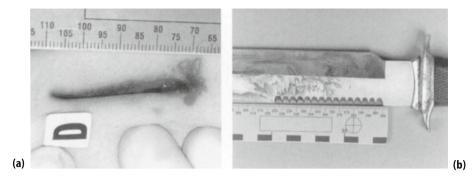
entry/exit or relative movement between the knife and body, although consideration should be given to the possibility of two separate thrusts.

Similarly, the margins may show "nicks" or separate cuts which imply relative movement between the victim and the knife while the blade was in the body – either twisting of the knife and/or movement of the victim. Close examination of the margins and underlying soft tissues may provide an early indication as to direction of thrust. If the edges appear similar then the direction is likely to have been nearly perpendicular to the skin surface but if the knife was inserted at an acute angle, one edge will be "shelved" (or bevelled) and may be identified by a ledge of soft tissue, with the opposite edge "undercut" (Fig 8.9).

The surrounding skin may show bruising or abrasion if the blade has been inserted up to its full length, i.e., up to the guard/handle, although this is a rare finding, occurring in 3–7% of cases and confined mainly to chest wounds (Figs 8.10 a, b)<sup>27,40</sup>. Spitz<sup>30</sup> suggested that bruising around the wound is most often caused by the fist holding the knife. As with cuts, a "tail" abrasion may be seen at one end of the wound where the cutting edge of the knife has run across the skin, probably on withdrawal. If the surrounding skin shows parallel superficial linear abrasions one should suspect a serrated blade as the weapon, but this is not a common finding and probably only occurs when the knife enters or exits at an angle, rather than perpendicular to the skin surface<sup>16</sup>.

#### Size

Wounds that lie parallel to the cleavage lines usually appear as slits but those that cross them tend to gape and the length of such wounds in their natural state – with the edges unapposed – will be an underestimate of the true size of the skin defect produced by the blade. A measurement with the edges apposed may provide some information about the width (distance between the two edges) of the blade. If the knife has been inserted and removed with the long axis of the blade perpendicular to the skin surface or with the long axis of the blade at an acute angle to the skin surface and the short axis (line across the width of the blade) parallel to the skin surface, then the wound length will usually correspond closely to the width of the blade – at that depth of penetration, i.e., distance from the tip. Occasionally, the length of the wound may be slightly less than the width of the blade due to elastic recoil of the skin but usually this is no more than a few millimetres.



**Fig. 8.10** (a). A stab wound on the chest showing an area of abrasion at one end, corresponding to the handle guard seen in Fig 8.10b. (b) The knife that was used to inflict the injuries seen in Figs 8.2 and 8.10a. Note the guard between the handle and the blade and the serrated edge.

However, all this assumes no movement of the knife in the wound or of the victim and in cases of multiple stab wounds very few fit the textbook description! It therefore follows that many different shapes and sizes can be produced by one knife, the length of the surface defect may often be greater than the width of the blade, and a variety of appearances does not necessarily indicate the use of more than one weapon.

# Internal Examination: Depth, Direction and Force Required

A careful, layered dissection of the soft tissues in conjunction with a full internal examination, exploring the track of each stab wound to its apparent termination and noting the organs/tissues penetrated, should allow the pathologist to make some comment on the depth and direction of each wound and the minimum force that would have been required. All three factors may assist the jury to decide whether the assailant intended to stab the victim. In addition, measurement of the size of the defects in the various layers may provide information about the weapon, particularly the taper of a blade.

## **Depth and Direction**

Unfortunately, there are anatomical constraints which often preclude the pathologist from being as precise as lawyers would wish. Obviously, the depth of a wound cannot be estimated if the point of maximum penetration cannot be determined. This may be because the tip of the blade entered a cavity, e.g., chest or abdomen, but did not injure an organ or injured a structure that may well have subsequently altered significantly in position or size, e.g., loops of mobile bowel or a collapsed lung. In these cases one may only be able to provide a minimum depth of penetration, often merely the thickness of the abdominal or chest wall. A more accurate estimate of depth may be obtained if the tip of the knife

terminated in bone, e.g., rib/spine, or a solid organ, e.g., liver; such a situation also facilitates determination of the direction of the wound track. There are, however, inherent inaccuracies in measuring the depth and direction of any wound even when the deepest point of penetration appears to have been identified. Probing of stab wounds prior to internal examination is not recommended as it is easy to produce false tracks; measurements of depth are therefore taken *after* dissection when the normal anatomical relations have been altered, e.g., skin/soft tissue flayed, ribcage removed.

The depth of a wound, or length of internal track, will in most cases be shorter than the length of the blade but in a significant minority the two measurements will approximate. Occasionally, however, the wound track will be longer, e.g., unsupported skin such as the anterior abdominal wall may indent on impact and therefore the wound depth may appear greater than the length of the blade if the depth is measured from the skin surface after it has recoiled to its normal position. The difference may be up to 5 cm and, as such, wound depth is of limited value in estimating the length of the blade<sup>40</sup>.

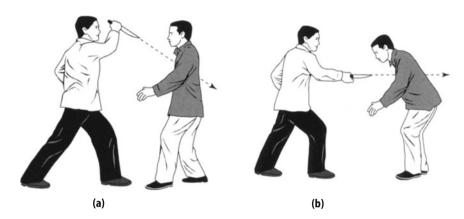
Autopsies are still traditionally performed with the deceased in a horizontal position, and this may not have been the victim's posture when the wounds were inflicted. Some organs, particularly the heart and liver, do alter position depending on whether the subject is erect or supine<sup>54</sup> and as a result, comment on the apparent depth and direction of the wound track should be accompanied by the term "approximate". Radiological studies have estimated the distances of various internal organs from the skin surface; the liver and spleen are most vulnerable, in some subjects these organs could be penetrated by a stab wound only 1 cm deep<sup>54,55</sup>, although even this figure does not allow for compression of the skin by the force of the impact.

Often, the surface characteristics, e.g., shelving/under-cutting, provide the best clue as to direction. Occasionally, a wound may be "through and through", i.e., one stab action produces an entry and exit wound, which obviously helps with estimating depth, and – if entry and exit can be identified – direction. In other cases, particularly where there are multiple wounds in a close grouping, it may be impossible to correlate surface wounds with the tracks of internal damage.

Although the size of the defects should be noted in each tissue layer and organ, some may accurately reflect the size of the blade, e.g., bone and serosal surfaces such as pleura and pericardial sac, while others, e.g., subcutaneous fat and muscle, may be readily distorted and difficult to measure.

The direction of the wound track is usually described in relation to three planes: sagittal, coronal and horizontal. It is customary to comment on whether the track goes forwards or backwards into the body, away from or towards an imaginary line running down the midline of the body, and upwards, downwards or close to the horizontal – the latter an imaginary line parallel to the ground, assuming the deceased in a standing position.

One should be cautious when interpreting the direction of the wound tracks, often there is more than one possible explanation. For example, a downward direction in a chest wound may be due to a downward thrust by the assailant with the victim in a vertical position or the impact of a blade held parallel to the ground with the torso of a crouching victim; the spine at an angle, rather than perpendicular, to the ground (Fig 8.11). It is probably better, therefore, to simply describe the direction – possibly aided by a diagram – and comment on whether it is consistent with the suggested scenarios.



**Fig. 8.11** The same angle of blade entry into the body may be caused by an overarm action **(a)** from the assailant with the victim vertical (left) or an underarm action **(b)** and the victim leaning forward (right).

The defendant's account may be extremely vague or immensely detailed and each legal team will attempt to get the pathologist to agree with their version of events – or at the very least to admit it was possible. Occasionally, counsel will utilize elaborate role-play in court in an effort to reconstruct the assault and although this may be good theatre it is potentially misleading. The pathologist should indicate the probable dynamic nature of an altercation, i.e., positions are likely to vary and unusual postures may be adopted. The victim and assailant will not necessarily be face to face, e.g., abdominal wounds may be more commonly inflicted from behind the victim<sup>36</sup>. In single stab wound homicides, the direction of the track of the wound may assume great significance; if the track extends straight back into the body, close to the horizontal, then a defence claim that the victim "ran onto the knife" or the assailant merely "prodded" the victim may be more readily accepted by a jury than if the direction is angled steeply upwards or downwards.

## **Force Required**

A case of fatal stabbing will almost inevitably involve the forensic pathologist being asked for an opinion on the force required to produce the injuries. This question still remains "almost impossible to answer" with any degree of objectivity. A detailed explanation would require a discourse on the biomechanics of stab wound infliction, involving relatively complicated mathematical formulae, often poorly recalled – or indeed understood – by forensic pathologists, most of whom are not known for their scientific rigour or fondness for physics. However, there are some relatively straightforward equations and concepts which should allow the pathologist to simplify matters for the jury.

Wounds occur when stresses on the body exceed the yield limit of the tissues. Any moving object has kinetic energy (E) measured in joules (J) and may be expressed in the equation  $E=1/2 \text{ mv}^2$ , where m is the mass (kg) and v the velocity (m/s) at impact. Therefore, the likelihood of injury is proportional to the mass of the knife and its velocity, or speed of approach. This formula would certainly apply with a thrown knife but things become more complicated in a stabbing action when one has to consider the work done by the assailant in moving the weapon

towards the victim. Energy is also described as "work done" and can be calculated by multiplying force measured in Newtons (N) by distance measured in metres (m). Newton's second law of motion states that force = mass x acceleration, and as acceleration is the rate of change in velocity, and momentum is a product of mass and velocity, it follows that force is equal to rate of change of momentum; dmv/dt (d = constant, m = mass, v = velocity, t = time), i.e., force is proportional to mass and velocity and inversely proportional to time. Finally, the area over which the energy is dissipated, i.e., energy density, is also an important factor; more damage occurring when it is concentrated over a small area.

Experimental studies on this subject date back to the nineteenth century but only recently has a degree of sophistication and objectivity been applied, largely due to superior technology improving methods of measurement. However, there remain significant problems; it is difficult in the current climate to obtain permission to stab cadavers, and animal models such as the pig are not identical to human tissue. Other materials, e.g., clay, have been used to simulate flesh but again are probably not quite like the real thing!

Knight<sup>56</sup> measured the forces required for penetration of cadavers and concluded that a load of as little as 0.5 kg was sufficient to penetrate abdominal skin using a "very sharply pointed knife", although measurements were made of a static load rather than a dynamic event. Skin was noted to be the soft tissue most resistant to penetration and sharpness of the tip of the knife was the most important factor affecting ease of penetration. It was stated that "once a knife penetrates the skin, no further force need be applied to cause rapid penetration of the subcutaneous tissues and any underlying organ, except for bone or calcified cartilage", which has often been misinterpreted as the assailant need not apply further force for deeper penetration. However, the experiment involved storage of energy in a spring and therefore for as long as that was greater than resistive forces, penetration would occur. Velocity, momentum and the shape of the blade were also felt to be important factors. Cadavers were positioned such that they leant forward onto volunteers holding a knife and penetration was possible if the knife was "firmly held in the operator's hand". This seemed to suggest that the "ran onto the knife" defence where the "assailant" stands still or is unaware of events while the victim impales him/herself on the knife - may be valid in some circumstances. Knight concluded that the hilt of the knife need not be supported upon a relatively rigid object, such as the hip of the holder, and he suggested the knife does not need to be held rigidly if it penetrates the skin rapidly<sup>57</sup>. Other authors have expressed scepticism; Spitz does not like casual holding of the knife by the assailant<sup>30</sup> and Purdue<sup>25</sup> suggests the object (knife) should be firmly fixed in the "ran onto the knife" scenario.

Green<sup>58</sup> inflicted wounds on cadavers from a distance of 15 cm and confirmed some of Knight's findings, in particular, the "small amount of pressure...required to penetrate the naked chest or abdomen". Green was of the opinion that it was difficult for an assailant to "hold back" once the skin had been breached. He found that larger knives required a greater force and short, thin-bladed knives were "ideal" weapons. The presence of clothing increased the thrust required and this was his explanation for the greater effort needed to remove the knife in many cases.

More recently, Jones et al.<sup>59</sup> stabbed pig tissue with two knives (one sharp, one blunt) at various speeds. A load cell between the handle and the blade measured the force and this was plotted against time to provide information on the forces required to penetrate tissues. Most resistance was offered by skin but the results contradicted earlier studies by finding significant secondary resistance in deeper

muscle layers. However, they provided no objective assessment of the sharpness of the knives and their "blunt" knife could not penetrate tissue, even at forces greater than 250 N. In addition, they found no correlation between speed of approach and force required for the sharp blade to breach the skin.

Other studies<sup>60,61</sup> have used motion analysis systems to assess human performance in stabbing actions, i.e., the ability to inflict a stab wound. They can also be criticised for not accurately reflecting real life, i.e., they do not reproduce the emotional element of an altercation, but they do provide objective measurements of impact velocity, energy and force. Miller and Jones<sup>60</sup> estimated the velocity (blade entry speed) of four stabbing actions; long overarm, short overarm, long underarm and short underarm. Values of up to 9.2 m/s were recorded for mean blade speed at entry and 11.4 m/s prior to entry. It should be no great surprise that "long" actions involved greater velocities because of extra speed generated over the longer acceleration paths. Therefore, the maximum speed generated is influenced by the manner in which the knife is held - or more accurately the stabbing action. Chadwick<sup>61</sup> persuaded 20 volunteers to execute three stabbing actions at near maximum effort: short thrust, horizontal sweep and overhand. Pulsed infrared light emitted from cameras and reflected back from markers placed on the subjects was detected by two or more cameras and used to measure velocity during the first (approach) phase, deriving values for energy and momentum. During the second (impact) phase an instrumented, force-measuring knife provided values for the four forces involved: axial (along the blade), cutting, lateral and torque (twisting). In terms of energy, the sweep action produced values greater than overhand and the thrust involved the least energy; the 95th percentile value was 69 J with a maximum of 103 J. In terms of force, the overhand action produced the largest, followed by the sweep then the thrust; the 95th percentile value for axial force was 1885 N, although forces greater than 2,000 N were measured. In addition, the force profile indicated two peaks, suggesting that other studies which had utilised drop-tower analysis (a knife falling vertically in a specially constructed tower), measuring only axial forces and showing only one force peak, may have shortcomings in terms of simulating blade penetration. Another study<sup>62</sup> using a device to determine the force - displacement behaviour of materials in high-impact loading situations, e.g., stabbing, measured impact velocities of up to 7.7 m/s, impact energies of up to 56 J and peak force in excess of 2,000 N.

Horsfall<sup>63</sup> asked volunteers to stab a target with underarm and overarm actions and used an instrumented knife to measure acceleration and axial force on the blade during impact, obtaining values for terminal velocity and load on contact to calculate the impact energy. The maximum energies were 64 J for underarm and 115 J for overarm, although the underarm motion was considered more difficult to defend. It was acknowledged that these figures may represent an overestimate as a hilt had been fitted to protect the volunteers hands, which possibly allowed them to generate more energy. The typical terminal velocities were in the range 6–10 m/s and loads on contact often approached 1,000 N. The authors stress that the energy measured is due to the kinetics of the knife and the stabbing action, *and* the muscular force (work) expended in penetration. A study<sup>64</sup> performed to assess the efficacy of stab-resistant body armour generated lower figures for energy; 42 J was considered representative of the "average" stab attempt.

It would seem, therefore, that there are three major components to be taken into account when deciding on the force required to produce penetration: characteristics of the knife, mechanics of the stabbing action, victim-related factors. Although

the latter is a matter for the pathologist, detailed comment on the force required may be limited by the absence of a weapon and/or a paucity of information on the stabbing action. With regard to the knife, mass, length and blade cross-sectional area appear relevant but sharpness of the tip is probably still the most important factor. O'Callaghan<sup>65</sup> assessed three methods of quantifying sharpness. He found that scanning electron microscopy – to measure the surface area of the tip – was the most accurate but it precluded further testing of the knife as the tip was sacrificed. Measuring the rate of compression into a standard material, e.g., modelling clay used for body armour testing, was less successful but slightly better than measuring the puncture resistance of layers of paper. Confirmation that the shape of the blade is an important factor in resistance to penetration came from finding that the force required decreased by an average of 68% when a military dagger was compared to a kitchen knife, the latter requiring greater force.

It has already been noted that the stabbing action, e.g., overarm, underarm, thrust or sweep, affects force and energy values and that the speed of approach (impact velocity) can approximate to 8–12 m/s.

As for victim-related factors, the presence and type of clothing may be relevant but in most cases the prime factor appears to be the target area, particularly the types of tissue injured and their resistance to penetration. O'Callaghan<sup>65</sup> performed experiments aimed to measure the forces generated in various stabbing scenarios and the resistance forces of different tissues. Human victims (protected by vests) ran, walked and stumbled onto an assailant holding a knife, first with their arm straight out and then with the forearm bent at 90° to the upper arm. Then the victim employed four different actions: standing still, walking, running, stumbling, while the assailant used three stabbing actions: light prodding, moderate force, large force. Peak forces were recorded for all scenarios. A separate experiment involved volunteers stabbing various human tissues with a kitchen knife and a military dagger; all noted penetration of the tissues "with little resistance", even the sternum. The results agreed with those of Knight<sup>56</sup> that only a small force – in O'Callaghan's study equivalent to about 2 kg - was required to obtain penetration with a sharp knife. The mean peak resistive forces were: 53 N for skin, 37 N for muscle and 1.75 N for fat. Interestingly, resistance of skin dropped to 30 N if the stab wound was parallel, rather than perpendicular, to cleavage lines. Cartilage resisted until 140 N and sternum until 200 N but neither offered a resistance force greater than 250 N. It was concluded that of the soft tissues, skin provided the greatest resistance to penetration but there were "considerable secondary resistance forces" in the underlying tissues. Only 60% of the initial force is required to continue penetration, and therefore to the assailant it may appear that the knife slips in easily but the application of force does not stop when the skin is penetrated. O'Callaghan confirmed that a "significant force" (about 60 N) is also required for knife removal. However, this was not due to clothing but (probably) to dynamic frictional forces of the tissues on the blade and the creation of a temporary cavity causing a partial vacuum.

All these simulated stabbing scenarios recorded peak forces in excess of 100 N, i.e., greater than the value required to penetrate skin and soft tissue, and in all the "stumble" or "ran onto the knife" situations the peak force was greater than or equal to 250 N, i.e., that required to penetrate the sternum. It was concluded that the common defence of "he ran onto the knife" may be perfectly valid from a scientific standpoint, even if the majority of jurors are highly sceptical. O'Callaghan acknowledged certain limitations in the studies and suggested further work on the

fracture mechanics of biological tissues and sharpness of the cutting edge as a factor. However, the results seem to indicate that penetration of skin and soft tissues is probably relatively easy with a sharp knife.

How does all this research assist the forensic pathologist in court and what general conclusions can be drawn? First, before commenting, the pathologist should be in possession of as much information as possible with regard to the three principal factors: weapon, alleged stabbing action, victim-specific factors, e.g., wounds and clothing. Or, if the information is incomplete – as in many cases it will be – the pathologist should stress the limitations or provisional nature of the conclusions.

Although the cutting edge, shape and length of the blade may be relevant it is generally agreed that sharpness of the tip is the most important characteristic to be taken into account when assessing ease of penetration. At present, there is no routinely used, scientific method of assessment; the pathologist has to resort to testing with the tip of a finger. The limitations and subjective nature of such an examination are obvious and the author favours simplicity, describing the tip as either blunt, moderately sharp or extremely sharp. Some lawyers, usually for dramatic effect, will ask a pathologist to examine the knife in court. If placed in such a position one should wear gloves and take care not to draw blood! If, however, this is a first examination of the knife, one should seek an adjournment in order to facilitate a proper assessment and comparison with the injuries.

The pathologist may be provided with a story regarding the alleged altercation, possibly involving the relative positions and actions of the "victim" and "assailant". Some scenarios can be incredibly elaborate, not to say downright fanciful, but it is the pathologist's duty to listen carefully and assess feasibility, using common sense and knowledge of the victim's injuries. It seems from experimental measurements of energy and forces involved that the victim can impale him/herself on the knife – including penetration of bone – with no movement from the "assailant", and therefore the defence that the victim "ran onto the knife" is possible in many single stab wound fatalities. It follows, therefore, that although the pathologist may describe a sharp force injury as a "stab wound" – because the internal depth is greater than the surface length – this does not necessarily mean that a stabbing action was employed by the alleged assailant.

Similarly, although wound depth may be used by lawyers to imply intent, given the ease with which sharp knives can penetrate tissues it is not necessarily safe to conclude that a deep wound was inflicted deliberately. Of greater importance is the structural damage to the victim, in particular, whether bone was penetrated. In general, bone provides greatest resistance followed by cartilage, skin and then muscle. Most victims are young and probably have relatively strong bones but older victims may have osteoporosis, facilitating penetration. As far as skin penetration is concerned, the age of the victim is irrelevant although the target site may be important; it has been suggested that stretched skin, such as on the chest between two ribs, is easier to pierce. In most cases, clothing will not provide any significant protection against a sharp knife although thick leather may occasionally impede penetration.

Finally, the pathologist can only really provide very broad categories to describe the force required – normally three: slight pressure, moderate force, severe force. However, extreme or considerable force may be appropriate when thick bone, e.g., the skull, has been penetrated. In the common scenario of a stab wound to the chest with a kitchen knife, a fractured rib (bone or calcified cartilage) is generally taken to indicate at least a moderate degree of force.

# **Examination of Clothing**

In an "ideal situation" the victim is found dead at the scene and an ambulance crew have not made efforts at resuscitation, which may include attempts to cut off the clothing. Unfortunately for the forensic pathologist, the victim is often pronounced dead in hospital having had some or all of their garments removed. In this case it is imperative that the clothing is retained by the police, and, if possible, statements taken from those who removed it stating the technique they employed. It will usually be examined by a forensic scientist for blood stains and defects but the pathologist should be informed of the type of clothing and the scientist's findings, and be prepared to personally view the items.

If the body is still at the scene, valuable evidence can be gathered by noting the pattern of blood staining on the clothing in relation to the position of the body. Consideration should be given to removing clothing – or other items such as ligatures – prior to transferring the body to the mortuary, as the latter will inevitably involve spread of blood which may obscure patterns and prints, e.g., of footwear.

Although it has been recommended that clothing should not be cut off the body so that artefactual defects are not confused with genuine assault-related damage, it does cause less blood-stain contamination than trying to pull a shirt or jersey off a body in full rigor mortis. Furthermore, forensic scientists can usually interpret cuts made to remove clothing. The best method of removal is still, therefore, a matter of debate; personal preference may be influenced by the individual factors in each case. Obviously, if clothing is cut, care needs to be taken to avoid pre-existing damage. Once removed, the items should be examined for defects, noting their size, shape and position – relative to seams/collar/midline. Whether issues of contamination will mean in future that this has to be performed in a sterile environment or performed after the forensic scientist's examination is a matter yet to be resolved.

The size and number of clothing defects does not always match the number of thrusts or the size and number of stab wounds. Due to folds in clothing, one stabbing action may cause more than one defect (Figs 8.12a, b) and in a bulky or loose item there may be damage to the clothing but no injury to the underlying skin. In contrast, a stab wound may occasionally be covered by undamaged clothing as a result of movement during an altercation, leaving the skin exposed at the moment of stabbing. Whether clothing has been damaged does not particularly assist in determining the manner of death although there is some truth in the traditional forensic teaching that suicide victims tend to pull clothing aside before inflicting injuries. Perforation of clothing is probably more common in homicide<sup>11,19</sup> but a significant number of suicidal stab wounds are inflicted through clothing; 28% of all cases<sup>3</sup> and 52% of those involving the trunk<sup>2</sup>. There may even be "hesitation" marks in clothing<sup>44</sup>. Therefore, damage to clothing does not necessarily imply homicide.

Examination of the clothing by a forensic scientist may be able to provide information on the implement and causation. It is apparently possible to tell a cut from a tear, a stab from a slash and whether the defect is "recent" or old<sup>66</sup>. Factors that determine the length of a stab cut in clothing include: degree of stretch of the fabric and whether it was taut or loose, sharpness of the knife blade, angle of the blade to the surface and to the weave of the fabric. Obviously, size of the weapon is also relevant but it has been demonstrated that dimensions in the clothing do not accurately reflect blade width<sup>67</sup>. In the majority of cases the cut in the garment measures

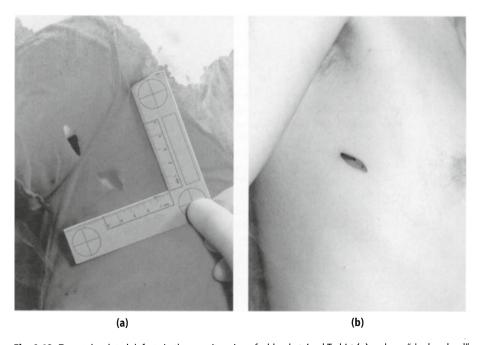
less than the width of the blade; the minority (7%) where the cut is larger, are thought to be due to the use of a blunt blade. Defects cannot be linked to a specific knife, but some fabrics may yield more information than others, i.e., "tightly knitted" seem better than "looser elastic" items<sup>68</sup>. Leather may also provide a more accurate reflection of blade characteristics.

Finally, knowledge of what the victim was wearing may influence the reply to a question posed about the degree of force required to cause a certain injury. Most items of clothing are probably easily penetrated by a sharp knife but several layers of leather may provide some resistance, particularly to a blunt knife.

## The Scene

If there is a "scene" then a visit by the pathologist may help determine the manner of death and address questions relating to position of the victim when stabbed and likelihood of blood contamination of the assailant.

Some cases, particularly involving single stab wounds or cuts to the neck, pose problems of interpretation and it may be difficult to differentiate homicide from suicide based on the pathology alone. Scene evidence may then be helpful. In suicide, one would expect the knife to be reasonably close to the body, unless it had been cleared away by a witness, although it should be remembered that victims can move significant distances after sustaining an injury that eventually turns out to be fatal. Karger's study<sup>2</sup> of sharp-force suicide, i.e., cutting and stabbing, found a



**Fig. 8.12** Two stab-related defects in the armpit region of a blood-stained T-shirt **(a)** and one "single-edged" stab wound in the corresponding region of the underlying chest **(b)**. The blunt end of the wound lies furthest from the nipple and nearest the armpit.

weapon "in situ" in 6 of 65 (9.2%) cases and only two cases of cadaveric spasm. Another study of 28 cases of self-stabbing found the knife within the body in 39% and nearby in the remainder<sup>3</sup>. The absence of a knife should therefore raise suspicion, as would an insecure scene and evidence of a disturbance. A note of intent may help resolve the issue but only a minority of suicide victims (7–28%) leave such a letter<sup>3,11,19</sup>.

Blood pattern analysis is a specialised field of forensic science but the pathologist should be able to draw some general conclusions and in any case should liaise with the forensic scientist regarding possible scenarios. Spray patterns, e.g., on the walls, may suggest arterial spurting while others, e.g., on the ceiling, may imply cast off from a knife. Swabs may be taken at the scene – including from the body – in an effort to identify DNA from an assailant, particularly if the suspect has a bleeding wound. Special attention should be paid to "drips" on the body unrelated to wounds.

# **Examination of Weapon**

A visit to the scene may provide the first opportunity to view a suspect weapon but in many homicides the knife is absent and some estimate of the weapon has to be made from the victim's injuries. If, however, a knife is provided for assessment, it is best to examine it *after* the autopsy – so that one can avoid an accusation of bias – and then compare wound and knife measurements. Because of possible contamination the weapon will probably be sealed in a plastic container, which may hamper accurate measurement and will normally preclude an assessment of the sharpness of the tip and cutting edge of the blade. Pathologists are therefore advised to be cautious with their comment on whether the knife could have produced certain wounds until detailed examination of the knife can be performed once the forensic scientists have finished taking prints and swabs.

The pathologist should make a note of various measurements and characteristics of the suspect weapon (Table 8.1). The aim is to determine whether it could have, or alternatively, could not have, caused the injuries on the victim. It is rare to be able to say that a particular weapon definitely caused an injury unless the tip of the knife broke off in the body and can be matched with the defective knife<sup>39,57</sup>. In the majority of cases one can only say that the injury was consistent with use of

**Table 8.1** Weapon examination and measurements.

- Type of knife
  - single or double-edged
  - serrated
- 2. Length of blade
- 3. Maximum width and taper of blade
- 4. Width of blade at same distance from tip as approximate depth of each stab wound
- 5. Thickness of blunt edge
- 6. Sharpness of tip
- 7. Sharpness of cutting edge
- 8. Nature and size of guard
- 9. Type and size of handle
- 10. Presence or absence of apparent blood stains on the blade
- 11. Weigh
- 12. Unusual distinguishing characteristics
- 13. Damage to weapon

the suspect weapon, although very occasionally comparison of the cutting edge of the knife with marks on the body, e.g., striations on rib cartilage<sup>69</sup> or parallel linear abrasions on the skin surface<sup>16</sup>, may provide compelling evidence. In such cases consideration should be given to retaining tissue for "tool mark analysis".

The relationships between type of knife and wound characteristics, e.g., shape, length, depth, have already been discussed. A comparison of the length of the blade with the approximate depth of each stab wound may allow the pathologist to comment on whether the blade was inserted to its full length. If this is likely and there is associated surface bruising or abrasion, then measurement of the size and shape of the portion of the handle adjacent to the blade will be important. It is useful to measure the width of the blade at a distance from the tip which corresponds to the approximate depth of each stab wound and compare it with the length of the surface wound. In most cases these measurements will either approximate or the wound will be larger but occasionally the blade width will be greater. As long as the disparity is not several centimetres this does not necessarily eliminate the blade as the weapon; indentation may occur, particularly of loose skin, and a blunt blade may not cut skin efficiently.

Estimation of the sharpness of the tip and cutting edge is of relevance to a comment on force required. Blood stains may be present on the blade but their absence does not preclude use of the weapon as a stabbing implement as the blade could have been cleaned in an attempt to eliminate trace evidence. Clothing may also "wipe" the knife but even where there has been no cleaning, a single stab wound may not necessarily leave visible blood stains on the blade, probably in part due to the rapidity of the action, leaving little time for blood to escape from severed vessels. The weight of the weapon may occasionally be relevant; in "chopping-type" injuries the appearances, e.g., bruised and abraded margins, underlying fractures (Fig 8.13), might suggest the use of a heavier weapon. Damage to the weapon, e.g., a broken or bent blade, may be related to metal strength and/or force employed. Finally, it has been suggested that photographs and a trace outline of the weapon may be useful for the pathologist's records<sup>70</sup>.

# Survival, Post-injury Activity and Mechanism of Death

#### **Survival**

When discussing survival there are two main questions to address. First, were the wounds necessarily fatal, or put another way, what were the chances of survival? This leads on to a consideration of prognostic factors and the reasons why some subjects survive their serious injuries and others die. Secondly, in fatal cases, how long was the survival period? This has implications for the extent of post-injury activity, and specifically, the ability to perform significant purposeful activity, which may be important if the deceased appears to have been found some distance from the location of the alleged incident.

Analysing the literature in an attempt to answer these questions is problematic as a comparison of studies is complicated by several factors<sup>71</sup>. Many of the larger studies come from the United States where survival figures relating to penetrating trauma include gunshot wounds, which in general have a higher mortality than stab wounds. Most studies are retrospective and many are based on a small number of cases.



**Fig. 8.13** An incised "chopping" wound over the front of the right shoulder showing a slice defect (arrow) on the head of the humerus. Injury also seen in Fig 8.5a and caused by the kukri in Fig 8.5b.

An assessment of the likelihood of surviving a stabbing assault can be greatly influenced by case selection<sup>72</sup>. For example, clinicians only see those that survive long enough to make it to hospital; many of their studies exclude subjects dying at the scene or on arrival at hospital and therefore the survival figures appear relatively high. Pathologists, on the other hand, only examine the dead and probably have an unduly pessimistic view of survival. Few studies look at all episodes (fatal and non-fatal) of sharp-force trauma although there are exceptions; Webb et al.<sup>33</sup> investigated 120 "knife assaults" in Edinburgh - all described as stabbings although some may have been slashes - and found that 17% were fatal. Trauma scoring was used to calculate the probability of survival and two cases were classified as "unexpected survival". Twelve of the 20 deaths were potentially survivable but they failed to reach hospital alive, emphasising the fact that the majority of deaths occur outside hospital and therefore a critical determinant of survival is the time taken to reach medical attention. Although there are likely to be geographical variations, it would seem that in Edinburgh at least, probably one in five of all stabbings turn out to be fatal. Of those that receive hospital treatment, the mortality figure for all penetrating trauma (in Scotland) is close to 8% (STAG, 2001, personal communication).

However, when one separates cases by site of injury a different picture emerges. Autopsy studies have consistently shown that stab injuries to the anterior trunk, in particular the chest, are common<sup>27,28,34,40,41,42</sup>. The chest is the site of the lethal wound in 75% of deaths due to multiple stab wounds and involvement of the heart occurs

in 58% of single stab wounds<sup>29</sup>. Stab wounds to the heart are therefore a major cause of death and this probably partly accounts for the many clinical studies detailing survival/mortality figures for such injuries. However, only a minority reach hospital alive. Differences between studies may be accounted for by variations in transport time and quality of paramedic treatment but it seems clear that the majority (60-90%) of those who receive a stab wound to the heart will die at the scene or "on arrival" at hospital<sup>73-77</sup>. The lay public (and press) are probably still amazed that anyone survives such an injury but we have now got to the stage where if the subject gets to hospital "alive", they probably have "a reasonably good chance of survival" Mortality figures in this small, select group range from 8% to 47% 79-89. The practical relevance to the pathologist is that anyone surviving to reach hospital with a cardiac stab wound can be considered lucky and are certainly in a minority. Survival figures are better than they used to be and the principal reasons are: decreased transport time to hospital (allowing faster diagnosis and treatment), improved fluid resuscitation, greater clinical skill - including the use of emergency thoracotomy. This form of intervention was popularised in the 1970s but is now performed less frequently, and generally only in those patients showing some signs of life, i.e., a pulse or blood pressure<sup>90</sup>. Thoracotomies performed in the operating room have a better survival rate than those performed in the emergency room<sup>43,71</sup> but even cases performed outside hospital are said to have a 10% chance of a successful outcome<sup>91,92</sup>.

In general terms, the prognosis in all cases of sharp-force trauma – homicide, accident and suicide – depends largely on three factors: time from wounding to skilled surgical intervention, severity or extent of injuries (multiple injuries are worse), physiological status on arrival at hospital; sinus rhythm and a recordable blood pressure are more favourable than asystole and/or unrecordable blood pressure. Death may occur from a potentially survivable wound because the victim did not receive prompt medical attention; either the episode of trauma was unwitnessed and the injury precluded the victim from obtaining help, or it occurred at an isolated location.

With particular reference to cardiac and chest stab wounds, the specific site of damage is relevant, i.e., great vessel (aorta, superior vena cava) and coronary artery involvement are consistently associated with a decreased survival although there is debate about whether injury to the left ventricle or right ventricle is more dangerous; some authors 13,87,89,93 claim that right-ventricle injury has a better prognosis, others<sup>57</sup> say that injury to the left ventricle may be less rapidly fatal, and others<sup>83</sup> have found no significant difference. Similarly, the occurrence of cardiac tamponade may be a favourable sign<sup>76,87,89,93</sup> although others disagree<sup>83</sup>. Associated abdominal injuries are believed to indicate a worse prognosis<sup>88</sup>, although on their own, stab wounds to the anterior abdominal wall are not particularly dangerous; of those that survive to hospital, approximately 50% show no significant organ injury and the mortality is about 1-3% 94,95. Small bowel, liver, stomach and colon are the structures most frequently injured; vascular injuries are less frequent but are associated with a higher mortality rate, especially vena caval wounds<sup>96-98</sup>. There is considerable debate over the optimum clinical management of abdominal stab wounds, largely regarding methods of diagnosing internal injuries and whether to perform an early laparotomy or adopt a "wait-and-see" approach 36,94,95,99. The latter may lead to criticism of medical staff in the event of a fatal outcome.

Stab wounds to the head are quite common in South Africa; morbidity and mortality are high, 30% develop vascular complications and the overall mortality – of

those that make it to hospital alive – is 26%<sup>37,100</sup>. Damage to an external carotid artery has been reported after an intra-oral stab wound<sup>101</sup> and somewhat surprisingly, full recovery – without neurological deficit – has been described following complete transection of a common carotid artery caused by a stab wound to the neck<sup>102</sup>. Spinal cord stab wounds are rare; even in South Africa where there is considerable experience, they account for only 0.24% of all treated stab wounds, despite being the cause of one quarter of all spinal cord injuries. The majority are the result of a single wound, 64% involve the thoracic spine, 30% involve the cervical segment and complete transection of the cord occurs in 21% of cases. Of those treated in hospital, the mortality rate is relatively low (3.6%); death is often attributed to meningitis, pulmonary embolus or renal failure and almost half occur after discharge from hospital<sup>103</sup>.

Whether alcohol intoxication acts as an independent prognostic factor is difficult to assess. Apart from the obvious problem of commenting on physical and mental effects during life merely from a single post-mortem blood alcohol concentration, many studies prevent conclusions from being drawn due to incomplete toxicology results. Nevertheless, reasonable suggestions may be made as to why alcohol might influence the outcome. It may affect the ability of the victim to defend himself and/or escape, impaired perception of the seriousness of the injury may limit the time available to call for help and there may be a reduced capacity to apply sufficient pressure to a bleeding point<sup>33</sup>. Accidents are more likely to occur if the victim is intoxicated as a result of drugs and/or alcohol and toxicology should therefore be an integral part of the autopsy. The results will probably not alter the medical cause of death but they may help to explain why the trauma occurred and why it proved fatal.

## **Post-injury Activity**

Questions may be asked regarding length of survival and the amount of post-injury activity that might have been possible. Pathologists should be wary of being too dogmatic; few injuries are necessarily immediately fatal and there are examples of "unusual, extended post-injury activity", such as ascending staircases or running several hundred metres, after receiving what turns out to be a fatal stab wound to the heart 41,42,57,104. The adrenal response presumably plays a part and there may be a delay before the victim realises they have been stabbed. It has been reported that one half of those who die from a single stab wound to the heart do so immediately, although in the same study, 22% of all sharp-force fatalities made some physical effort after injury<sup>41</sup>. Karger<sup>105</sup> studied eight cases in which suicidal stab wounds had penetrated the heart, and of these, four showed "physical activity" for between two and ten minutes; the larger cardiac wounds were associated with less activity. Rarely, survival may extend to several hours<sup>106</sup>. Indeed, the first successful operation on a penetrating cardiac stab wound, performed by Ludwig Rehn in 1896<sup>107</sup>, occurred almost two days after the right ventricle was injured. Levy and Rao<sup>42</sup> reported that 71% of the 22 stab wound victims they studied survived for greater than five minutes, which they attributed to a marked improvement in trauma unit response times and more aggressive medical treatment. Di Maio<sup>24</sup> states that postinjury physical activity (and by implication survival period) depends on four factors: vascularity of organ pierced, size of blood vessel severed, amount of blood lost, rapidity of blood loss. It is generally accepted that in most circumstances injuries to the heart and great vessels will be more rapidly fatal than injuries to

other organs such as the lung or liver and that bleeding is more brisk from arteries than veins. Finally, multiplicity of wounds is a relevant factor, the larger the number the less likely there is to be significant post-injury activity. It therefore behoves the pathologist to be cautious when asked to comment on these matters; assess the type, number and severity of the injuries, consider the age and health of the victim, listen to the suggested scenarios and be ready to admit that extensive penetrating injuries – even to the heart – are not necessarily immediately fatal and do not preclude a victim from making some physical movements.

#### Mechanism of Death

The causes and mechanisms of death in fatal sharp-force trauma are generally more straightforward and easier to explain to a jury than those due to blunt-force trauma. Deaths can be described as "early" or "late" and most of the former are due to blood loss, external or internal, e.g., haemothorax, haemopericardium. However, wounds to the neck can open the trachea, allowing blood to percolate down - and obstruct - the airways. Such wounds may also open the jugular veins, permitting air entry and embolisation to the heart. This may be detected on a pre-autopsy chest x-ray and although some authors recommend opening the heart under water, the finding of abundant froth in the heart on routine slicing would normally be sufficient to diagnose air embolus. It is probably underestimated clinically; apparently aspiration of air from the ventricles can be of benefit during resuscitation of victims with cardiac and lung wounds<sup>43</sup>. Late deaths are those that occur after the initial period of resuscitation and treatment, often within a few days but sometimes weeks, months or even years later. In the early part of this period, infection, e.g., peritonitis, meningitis, is the principal risk although vascular occlusion due to thrombosis and/or embolism does occur. In such cases the issue of causation may be questioned; it could be argued that an infection was "picked up in hospital" or caused by medical staff. This can usually be countered by the claim that the victim would not have been in hospital had he/she not been assaulted. In reality, the defence would be unlikely to succeed with this approach but clinicians should be aware that their actions and notes may come under considerable scrutiny and therefore both should be of the highest quality.

Complications can arise in patients treated for penetrating cardiac injuries<sup>84,85,88</sup>. Initially, hypotension, multiple transfusions and cerebral oedema can lead to coagulopathy, sepsis, shock, cardiac arrhythmias, myocardial infarction, and cerebral complications, e.g., anoxic encephalopathy and paraplegia. These problems may ultimately be responsible for deaths on the intensive treatment unit (ITU) as a result of multi-system organ failure, adult respiratory distress syndrome (ARDS) or brain death. In the heart, valvular damage and intracardiac shunts may require urgent surgical repair. Other vascular complications, e.g., aneurysms and arterial-venous fistulas, may present in the longer term 37,100,108-111. It is important, therefore, for the pathologist to bear in mind that such cases may present as a "natural death" and one needs to consider the possibility that an episode of trauma, maybe many years previously, might be the underlying cause. Kennedy<sup>112</sup> described a case of bowel obstruction due to herniation through a defect in the diaphragm caused by a self-inflicted stab wound three years previously. The patient survived but it is not difficult to envisage fatalities in such circumstances.

## **Practical Tips**

Table 8.2 amounts to a distillation of practical advice on matters relating to the performance of a sharp-force trauma autopsy and associated events before and after the examination. It is not a comprehensive list of what to do but merely a selection of tips the author considers may improve the quality of the report and/or assist understanding by lawyers and the jury, hopefully aiding presentation of the pathology evidence in court. Some of the suggestions are specific to stab wounds, other points are applicable to all "homicide" autopsies and of course, not all the advice is relevant in any one case. To the experienced forensic pathologist much of this will be obvious and while some recommendations reflect "best practice", other issues, e.g., layout and style of report, are a matter of personal preference.

If medical/surgical intervention occurred then the clinical notes should be read prior to the autopsy. Useful information can be obtained, particularly regarding resuscitation attempts: chest compression, amount of intravenous fluid administered, operations performed, e.g., thoracotomy, which may help to explain some "injuries" and avoid misinterpretation. An unsutured chest drain incision can resemble a stab wound, as often the drain has been removed in the process of "tidying up" by nursing staff. The latter practice should be discouraged but habits which are applicable to the vast majority of hospital patients are difficult to change for the relatively small number of suspicious trauma deaths seen in UK hospitals. Similarly, a surgeon may decide it is neater to incorporate the stab wound into the thoracotomy incision. This exasperates pathologists, as obviously it obliterates any external evidence of the wound size or shape and in many cases it would appear to have been easier to avoid the stab wound. However, it is recognised that our clinical colleagues have different (life-saving) priorities and a discussion with the surgeon will usually clear up any doubt as to what was done. Occasionally, it may be necessary to have the surgeon present at the autopsy.

**Table 8.2** Practical tips.

Pre-autopsy	Read hospital notes Discuss with surgeon Advise police to recover hospital samples Consider x-ray
Autopsy	Examine clothing Use adhesive lettered labels to identify wounds Use transparent adhesive tape to approximate wound margins Photograph each stab wound with scale Consider retaining tissue (penetrated cartilage) for "tool mark analysis"
Post-autopsy	Examine weapon Attend "defence" autopsy Consider experiment to assess validity of "unusual" scenario Refer clinical matters to an appropriate specialist
Report	Use lay terms Use imperial and metric units Describe external and internal injuries together for each stab wound Consider diagrams

The administration of intravenous fluids may significantly alter blood concentrations of drugs and alcohol, hampering assessment of post-mortem samples, and therefore, ideally, "pre-transfusion" samples should be analysed. The police may need a reminder to seize these, usually from biochemistry and/or haematology. They should not delay as many departments discard samples after a short time, e.g., two days. Finally, before the autopsy, consideration should be given to x-raying the body (or part thereof) if there is a likelihood of air embolus or retained radio-opaque fragments, e.g., glass or metal from a knife blade. X-rays should help to localise the fragment, enabling retrieval and avoidance of injury to the pathologist.

It is best practice to examine the clothing for defects and if items have been removed in hospital they should be recovered by the police. The photographic identification of surface wounds and their subsequent presentation to a jury, particularly where there are multiple stab wounds close together, may be helped by the application of an adhesive label (bearing a letter) to the skin adjacent to each wound (Fig. 8.14).

Photographs indicating the general disposition of injuries and their relationship to each other should be taken, followed by close-up photographs of each wound, with and without a scale; transparent adhesive tape placed across the wound can assist photography and measurement of wound length by approximating the margins. A photographic record is important; it corroborates a description of the size and shape of the wounds and helps to ensure that injuries are described in a report "on the correct side" – it is easy to get it wrong in the notes. Some pathologists mark the injuries on body diagrams but these are probably unnecessary if good-quality photographs are available and full notes are made, although a sketch



Fig. 8.14 Adhesive labels applied to the skin to assist identification of multiple stab wounds.

diagram or instant photograph may assist in tracing the tracks of the labelled wounds once the skin has been reflected.

Suspect weapons should be examined – as detailed above – *after* the autopsy. It is advisable to attend the examination performed by the "defence" pathologist as this facilitates exchange of relevant information and early identification of points of disagreement, which can ultimately save time for the court. By the very nature of the first examination, appearances at a subsequent autopsy are not the same, e.g., volumes of blood and their location will not be apparent and the direction of stab wound tracks may be even more difficult to assess after evisceration of the internal organs. It may be potentially misleading if the "defence" pathologist does not have information that was available to the "prosecution" pathologist. Another reason for attending is that occasionally things will be missed at the first autopsy – even by the best pathologists – and it is wise to be aware of any significant additional findings rather than be "ambushed" in court.

Many cases are straightforward but occasionally a scenario is put to the pathologist that is slightly unusual and may seem almost unbelievable. However, one should be careful of dismissing the hypothesis out of hand without supporting evidence. O'Callaghan et al.<sup>35</sup> reported an experiment which showed that a glass shard could be used as a stabbing weapon without the assailant sustaining injury to his hand; an "expert" medical witness had previously opined that the hand would be injured in such an action. Although there are significant difficulties in recreating the perfect experimental model, it is worth considering whether reconstruction of the alleged incident, particularly with an objective measurement of forces, might enable the "expert witness" to provide a more informed (and accurate) opinion<sup>31,113</sup>.

The pathologist will sometimes be asked to comment on medical and surgical matters, particularly related to questions about whether clinical intervention contributed to the death or whether the injuries were necessarily fatal. Other than providing the court with general guidance, the pathologist should be careful not to stray out of his/her personal field of expertise. If "clinical" issues are likely to be raised, pathologists should advise the legal authorities to seek an appropriate specialist opinion, e.g., medical personnel who regularly examine trauma patients (live and dying), such as A & E staff, may be able to offer a more accurate opinion on chances of survival, using trauma scoring systems<sup>114</sup>.

The report should be easy to understand. It may be a difficult balancing act to make the report appear as if it were written by a professional, utilising terminology that is recognised by the general public, and yet does not read like an episode of "Noddy performs an autopsy". However, the experts who come across well in court are those who use colloquial language, e.g., belly button and breastbone rather than umbilicus and sternum. It is therefore advisable to use sufficient lay terminology to make the report readable but not too much that it detracts from the professional nature of the statement. When writing a report it should be borne in mind that many people still think in feet and inches and it will save time and endear you to the judge - a tactic not to be underestimated - if imperial and metric measurements are provided, particularly for wound depths. It also facilitates presentation in court if the external appearances and internal track of each wound are described together rather than the traditional separation of external and internal injuries under different headings. There seems to be a move away from showing photographs in court, even prosecution lawyers are increasingly reluctant to subject the jury to what can be horrific sights, and as such, computer-generated diagrams of bodies or skeletons may be used to portray the number and distribution of injuries in a more aesthetically pleasing way. Diagrams may also help to explain the direction of stab wound tracks and obviate the need for the pathologist to demonstrate them personally, either on him/herself or a court official.

#### **Questions to Address**

During the investigation of a death due to incised wounds the pathologist will be asked a variety of questions by representatives of several different "interested parties" or agencies. Initially, the police will want guidance on the likely manner of death, scaling down their investigation should it be an accident or suicide. In such cases the relatives of the deceased, insurance companies and legal officials may ask questions at an inquest/inquiry. In cases of homicide which reach court, prosecuting and defence counsel will usually wish to examine the "expert witness" and the judge may also pose questions.

Some of these queries are impossible to answer and it is the duty of the forensic pathologist to inform the court of the limits of the pathology evidence<sup>70</sup>. For example, the autopsy findings alone cannot identify the age, sex, height or handedness of an assailant, the number of assailants or the order of infliction of the wounds. Intent, i.e., what was going through the assailant's mind at the time of the assault, is not a matter for the pathologist. Some questions may be best left for others to answer, e.g., manner of death and likelihood of survival, but it will do no harm for the pathologist to consider them. Table 8.3 brings together many of the issues discussed in the preceding sections of this chapter. It is intended as an aidememoire of the points to bear in mind when constructing the "Comment" section of a report, although it may be that some questions are best answered in court after

Table 8.3 Points to consider including in the comment section of an autopsy report.

Wounds	number
wounds	type
	- stab/cut
	- "defence"
	- others, e.g., blunt force, pressure to neck
	distribution
	depth
	direction (of stab wounds)
	structures damaged
	force required
	the "fatal" injuries
	the latar injunes
Mechanism of death	
Manner of death	
Cause of death	
Possible contributory factors	drugs/alcohol
,	natural disease
	surgical intervention
14/	Associated the second of the s
Weapon	type
	size
	sharpness of blade
Post-injury activity	
Likelihood of survival	
Entermoda di Salvival	

due consideration of all the available evidence. Many of the points are purely factual, others require the expression of an opinion and rarely will they all be relevant in any one case. Where an opinion is required, one should remember that, as with many aspects of forensic pathology, caution is advised and over-interpretation is not to be recommended.

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#### References

- 1. The New Shorter Oxford English Dictionary. Oxford: Clarendon Press, 1993.
- Karger B, Niemeyer J, Brinkmann B. Suicides by sharp force: typical and atypical features. Int J Legal Med 2000;113:259–62.
- 3. Start RD, Milroy CM, Green MA. Suicide by self-stabbing. For Sci Int 1992;56:89-94.
- 4. Karger B, Rothschild MA, Pfeiffer H. Accidental sharp force fatalities beware of architectural glass, not knives. For Sci Int 2001;123:135–9.
- 5. Criminal Statistics England and Wales, 1999. HMSO.
- Office for National Statistics, Mortality Statistics. Review of the Registrar General on Deaths in England and Wales, 1998 (Table 7. Death rates per 1,000,000 population). London: The Stationery Office.
- Scottish Executive Central Research Unit (1999), Homicide in Britain. Edinburgh: The Stationery Office.
- 8. Scottish Executive (1999) Statistical Bulletin. Criminal Justice Series. Homicide in Scotland 1998. Government Statistical Service.
- Bleetman A, Perry CH, Crawford R, Swann IJ. Effect of Strathclyde Police initiative "Operation Blade" on accident and emergency attendances due to assault. J Accid Emerg Med 1997;14:153–6.
- 10. Karlsson T. Sharp-force homicides in the Stockholm area, 1983-1992. For Sci Int 1998;94:129-39.
- 11. Karlsson T, Ormstad K, Rajs J. Patterns in sharp force fatalities a comprehensive forensic medical study: Part 2. Suicidal sharp force injury in the Stockholm area, 1972–1984. J Forensic Sci 1988;33:448–61.
- 12. Watanabe T, Kobayashi Y, Hata S. Harakiri and suicide by sharp instruments in Japan. Forensic Sci 1973;2:191–9.
- 13. Byard RW, Klitte A, Gilbert JD, James RA. Clinicopathologic features of fatal self-inflicted incised and stab wounds. A 20-year study. Am J For Med Path 2002;23:15–18.
- 14. Sperry K, Campbell HR. An elliptical incised wound of the breast misinterpreted as a bite injury. J Forensic Sci 1990;35:1226–35.
- 15. Milroy CM, Rutty GN. If a wound is "neatly incised" it is not a laceration. BMJ (letter) 1997;315:1312.
- Ciallella C, Caringi C, Aromatario M. Wounds inflicted by survival knives. For Sci Int 2002;126:82-7.
- 17. Shepherd JP, Shapland M, Pearce NX, Scully C. Pattern, severity and aetiology of injuries in victims of assault. J Roy Soc Med 1990;83:75–8.
- 18. Shepherd JP, Price M, Shenfine P. Glass abuse and urban licensed premises. J Roy Soc Med (letter) 1990:83;276–7.
- 19. Karlsson T. Homicidal and suicidal sharp force fatalities in Stockholm, Sweden. Orientation of entrance wounds in stabs gives information in the classification. For Sci Int 1998;93:21–32.
- 20. Kuroda N, Saito K, Takada A, Watanabe H, et al., Suicide by self-stabbing in the city of Tokyo a review of accumulated data from 1976 to 1995. Jap J Leg Med 1997;51:301–6.

- 21. Di Nunno N, Costantinides F, Bernasconi P, Di Nunno C. Suicide by hara-kiri. A series of four cases. Am J For Med Path 2001;22:68–72.
- 22. Betz P, Tutsch-Bauer E, Eisenmenger W. "Tentative" injuries in a homicide. Am J For Med Path 1995;16:246–8.
- 23. Herbst J, Haffner H-Th. Tentative injuries to exposed skin in a homicide case. For Sci Int 1999;102:193-6.
- 24. Di Maio DJ, Di Maio VJM (Eds). Forensic pathology. New York: Elsevier, 1989.
- 25. Purdue BN. Cutting and piercing wounds. In: Mason JK, Purdue BN, editors. The pathology of trauma, 3rd edn. London: Arnold, 2000.
- 26. Katkici U, Ozkok MS, Orsal M. An autopsy evaluation of defence wounds in 195 homicidal deaths due to stabbing. J Forensic Sci Soc 1994;34:237–40.
- 27. Hunt AC, Cowling RJ. Murder by stabbing. For Sci Int 1991;52:107–12.
- 28. Rogde S, Hougen H.P, Poulsen K. Homicide by sharp force in two Scandinavian capitals. For Sci Int 2000;109:135–45.
- 29. Rouse DA. Patterns of stab wounds: a six-year study. Med Sci Law 1994;34:67-71.
- Spitz WU. Sharp force injury. In:Spitz WU, editor. Medicolegal investigation of death, 3rd edn. Illinois: CC Thomas, 1993, Ch. VIII.
- 31. Hirt M, Karger B. Fatal brain injury caused by the free-flying blade of a knife case report and evaluation of the unusual weapon. Int J Legal Med 1999;112:313–14.
- 32. Rothschild MA, Karger B, Schneider V. Puncture wounds caused by glass mistaken for stab wounds with a knife. For Sci Int 2001;121:161-5.
- 33. Webb E, Wyatt JP, Henry J, Busuttil A. A comparison of fatal with non-fatal knife injuries in Edinburgh. For Sci Int 1999;99:179–87.
- 34. Ormstad K, Karlsson T, Enkler L, Law B, Rajs J. Patterns in sharp force fatalities a comprehensive forensic medical study. J Forensic Sci 1986;31:529-42.
- 35. O'Callaghan PT, Jones MD, James DS, Leadbeatter S, Evans SL, Nokes LDM. A biomechanical reconstruction of a wound caused by a glass shard a case report. For Sci Int 2001;117: 221–31.
- 36. Lambrianides AL, Rosin RD. Penetrating stab injuries of the chest and abdomen. Injury 1984;15:300-3.
- 37. Kieck CF, De Villiers JC. Vascular lesions due to transcranial stab wounds. J Neurosurg 1984;60:42-6.
- 38. Deb S, Acosta J, Bridgeman A, Wang D, Kennedy S, Rhee P. Stab Wounds to the head with intracranial penetration. J Trauma: Injury, Infection and Critical Care 2000;48:1159-62.
- 39. Lettington  $\bar{W}$ . Stabbing to right temple with partial knife blade retention: differentiating stab from slash wound. J Clin For Med 2000;7:45–7.
- 40. Murray LA, Green MA. Hilts and knives: a survey of ten years of fatal stabbings. Med Sci Law 1987;27:182-3.
- 41. Thoresen SO, Rognum TO. Survival time and acting capability after fatal injury by sharp weapons. For Sci Int 1986;31:181-7.
- 42. Levy V, Rao VJ. Survival time in gunshot and stab wound victims. Am J For Med Path 1988;9:215-17.
- 43. Demetriades D. Cardiac wounds. Experience with 70 patients. Ann Surg 1986;203:315-17.
- 44. West I. Single suicidal stab wounds a study of three cases. Med Sci Law 1981;21:198-201.
- 45. Gojanovic MD. Homosexual homicides. J Clin For Med 1998;5:191-4.
- 46. Bell MD, Vila RI. Homicide in homosexual victims. Am J For Med Path 1996;17:65-9.
- 47. R v Cook (1982) Crim LR 670.
- 48. Byard RW, Gilbert JD, James RA. Traditional punishment and unexpected death in Central Australia. Am J For Med Path 2001;22:92–5.
- 49. Boyle EM, Maier RV, Salazar JD, et al. Diagnosis of injuries after stab wounds to the back and flank. J Trauma: Injury, Infection & Critical Care 1997;42:260–5.
- 50. Ivatury RR, Prakashchandra MR, Nallathambi M, Gaudino J, Stahl WM. Penetrating gluteal injuries. J Trauma 1982;22:706–9.
- 51. Mercer DW, Buckman RF, Sood R, Kerr TM, Gelman J. Anatomic considerations in penetrating gluteal wounds. Arch Surg 1992;127:407–10.
- 52. Fekete JF, Fox AD. Successful suicide by self-inflicted multiple stab wounds of the skull, abdomen and chest. J Forensic Sci 1980;25:634–7.
- 53. Cox HT. The cleavage lines of the skin. Br J Surg 1941;29:234-40.
- 54. Bleetman A, Dyer J. Ultrasound assessment of the vulnerability of the internal organs to stabbing: determining safety standards for stab-resistant body armour. Injury, Int J Care Injured 2000;31:609–12.

55. Connor SE, Bleetman A, Duddy MJ. Safety standards for stab-resistant body armour: a computer tomographic assessment of organ to skin distances. Injury 1998;29:297–9.

- 56. Knight B. The dynamics of stab wounds. Forensic Science 1975;6:249-55.
- 57. Knight B (ed). Forensic pathology, 2nd edn. London: Arnold, 1996.
- 58. Green MA. Stab wound dynamics a recording technique for use in medico-legal investigations. J Forensic Sci Soc 1978;18:161–3.
- 59. Jones S, Nokes L, Leadbeatter S. The mechanics of stab wounding. For Sci Int 1994;67:59-63.
- 60. Miller SA, Jones MD. Kinematics of four methods of stabbing: a preliminary study. For Sci Int 1996;82:183–90.
- 61. Chadwick EKJ, Nicol AC, Lane JV, Gray TGF. Biomechanics of knife stab attacks. For Sci Int 1999;105:35–44.
- 62. Chadwick EKJ, Nicol AC, Floyd S, Gray TGF. A telemetry-based device to determine the force-displacement behaviour of materials in high-impact loading situations. J Biomechanics 2000;33:361–5.
- 63. Horsfall I, Prosser PD, Watson CH, Champion SM. An assessment of human performance in stabbing. For Sci Int 1999;102:79–89.
- PSDB Stab-resistant body armour test procedure. Home Office Science and Technology Group, 1993.
- 65. O'Callaghan PT. The biomechanics of stab wounds. Ph.D. Thesis. Cardiff University, 1999.
- 66. Monahan DL, Harding HWJ. Damage to clothing cuts and tears. J Forensic Sci 1990;35:901-12.
- 67. Costello PA, Lawton ME. Do stab cuts reflect the weapon which made them? J Forensic Sci Soc 1990;30:89–95.
- 68. Taupin JM. Comparing the alleged weapon with damage to clothing the value of multiple layers and fabrics. J Forensic Sci 1999;44:205–7.
- Rao VJ, Hart R. Tool mark determination in cartilage of stabbing victim. J Forensic Sci 1983;28:794–9.
- 70. Taff ML, Boglioli LR. Science and politics of cutting and stabbing injuries in the USA. J Clin For Med 1998;5:80–4.
- 71. Asensio JA, Murray J, Demetriades D, et al. Penetrating cardiac injuries: A prospective study of variables predicting outcomes. J Am Coll Surg 1998;186:24–34.
- 72. Trinkle JK. Penetrating heart wounds: Difficulty in evaluating clinical series. Ann Thorac Surg 1984;38:181-2.
- 73. Demetriades D, van der Veen BW. Penetrating injuries of the heart: Experience over two years in South Africa. J Trauma 1983;23:1034-41.
- 74. Sugg WL, Rea WJ, Ecker RR, Webb WR, Rose EF, Shaw RR. Penetrating wounds of the heart. An analysis of 459 cases. J Thorac Cardiovasc Surg 1968;56:531–45.
- 75. Oakland C, Vivien J. Penetrating cardiac injuries. BMJ (letter) 1987;295:502.
- 76. Campbell NC, Thomson SR, Muckart DJJ, Meumann CM, van Middelkoop I, Botha JBC. Review of 1,198 cases of penetrating cardiac trauma. Br J Surg 1997;84:1737–40.
- 77. Naughton MJ, Brissie RM, Bessey PQ, McEachern MM, Donald JM, Laws HL. Demography of penetrating cardiac trauma. Ann Surg 1989;209:676-81.
- 78. Henderson VJ, Smith S, Fry WR, et al. Cardiac Injuries: Analysis of an unselected series of 251 cases. J Trauma 1994;36:341–8.
- 79. Baker JM, Battistella FD, Kraut E, Owings JT, Follette DM. Use of cardiopulmonary bypass to salvage patients with multiple-chamber heart wounds. Arch Surg 1998;133:855–60.
- 80. Thourani VH, Feliciano DV, Cooper WA, et al. Penetrating cardiac trauma at an urban trauma center: A 22-year perspective. Am Surgeon 1999;65:811–18.
- 81. Velmahos GC, Degiannis E, Souter I, Saadia R. Penetrating trauma to the heart: A relatively innocent injury. Surgery 1994;115:694–7.
- 82. Marshall WG, Bell JL, Kouchoukos NT. Penetrating cardiac trauma. J Trauma 1984;24: 147-9.
- 83. Asensio JA, Berne JD, Demetriades D, et al. One hundred five penetrating cardiac injuries: A 2-year prospective evaluation. J Trauma: Injury, Infection & Critical Care 1998; 44:1073–82.
- 84. Ivatury RR, Rohman M, Steichen FM, Gundez Y, Nallathambi M, Stahl WM. Penetrating cardiac injuries: Twenty-year experience. Am Surgeon. 1987;53:310–17.
- 85. Attar S, Suter CM, Hankins JR, Sequeira A, McLaughlin JS. Penetrating cardiac injuries. Ann Thorac Surg 1991;51:711–16.
- 86. Harris DG, Papagiannopoulos KA, Pretorius J, Van Rooyen T, Rossouw GJ. Current evaluation of cardiac stab wounds. Ann Thorac Surg 1999;68:2119–22.
- 87. Arreola-Risa C, Rhee P, Boyle EM, Maier RV, Jurkovich GG, Foy HM. Factors influencing outcome in stab wounds of the heart. Am J Surg 1995;169:553-6.

- 88. Mittal V, McAleese P, Young S, Cohen M. Penetrating cardiac injuries. Am Surgeon 1999; 65:444-8.
- 89. Tyburski JG, Astra L, Wilson RF, Dente C, Steffes C. Factors affecting prognosis with penetrating wounds of the heart. J Trauma: Injury, Infection & Critical Care 2000;48:587-91.
- 90. Brown SE, Gomez GA, Jacobson LE, Scherer T, McMillan RA. Penetrating chest trauma: Should indications for emergency room thoracotomy be limited? Am Surgeon 1996;62:530–4.
- 91. Craig R, Clarke K, Coats TJ. On-scene thoracotomy: a case report. Resuscitation 1999;40:45-7.
- 92. Coats TJ, Keogh S, Clark H, Neal M. Prehospital resuscitative thoracotomy for cardiac arrest after penetrating trauma: Rationale and case series. J Trauma: Injury, Infection & Critical Care 2001;50:670-3.
- 93. Moreno C, Moore EE, Majure JA, Hopeman AR. Pericardial tamponade: A critical determinant for survival following penetrating cardiac wounds. J Trauma 1986;26:821-5.
- 94. Leppaniemi AK, Voutilainen PE, Haapiainen RK. Indications for early mandatory laparotomy in abdominal stab wounds. Br J Surg 1999;86:76–80.
- 95. Lee WC, Uddo JF, Nance FC. Surgical judgement in the management of abdominal stab wounds. Ann Surg 1984;199:549–54.
- 96. Asensio JA, Chahwan S, Hanpeter D, et al. Operative management and outcome of 302 abdominal vascular injuries. Am J Surg 2000;180:528-34.
- 97. Byrne DE, Pass HI, Crawford FA. Traumatic vena caval injuries. Am J Surg 1980;140:600-2.
- 98. Starzl TE, Kaupp HA, Beheler EM, Freeark RJ. The treatment of penetrating wounds of the inferior vena cava. Surgery 1962;51:195-204.
- 99. Goldberger JH, Bernstein DM, Rodman GH, Suarez CA. Selection of patients with abdominal stab wounds for laparotomy. J Trauma 1982;22:476–80.
- 100. du Trevou MD, van Dellen JR. Penetrating stab wounds to the brain: The timing of angiography in patients presenting with the weapon already removed. Neurosurgery 1992;31:905–11.
- 101. Hodges C, Medberry C, Geehan D. Laceration of the external carotid artery after an intraoral stab wound. J Trauma: Injury, Infection and Critical Care 1998;45:644-6.
- 102. Mehanna HM, MacGregor FB. The case of the missing carotid artery a well aimed knife! J Laryngol Otol 1998;112:387–8.
- 103. Peacock WJ, Shrosbree RD, Key AG. A review of 450 stab wounds of the spinal cord. S Afr Med J 1977;51:961-4.
- 104. Spitz WU, Petty CS, Fisher RS. Physical activity until collapse following fatal injury by firearms and sharp pointed weapons. J Forensic Sci 1961;6:290–300.
- 105. Karger B, Niemeyer J, Brinkmann B. Physical activity following fatal injury from sharp pointed weapons. Int J Leg Med 1999;112:188-91.
- 106. Perdekamp MG, Riede U-N, Pollak S. Penetrating stab wound of the heart with extraordinarily long survival time. Arch Kriminol 2000;206:102–9.
- 107. Blatchford JW. Ludwig Rehn: The first successful cardiorrhaphy. Ann Thorac Surg 1985;39:492-5.
- 108. Nathoo N, Nadvi SS. Traumatic intracranial aneurysms following penetrating stab wounds to the head: two unusual cases and review of the literature. Cent Afr J Med 1999;45:213–17.
- 109. Cha EK, Mittal V, Allaben RD. Delayed sequelae of penetrating cardiac injury. Arch Surg 1993;128:836-41.
- 110. Chukwuemeka A, Curry P, O'Riordan J. False aneurysm of the left ventricle after a stab wound to the chest. Ann R Coll Surg Engl 1999:81:94–6.
- 111. Maitre B, Jouveshomme S, Isnard R, Riquet M, Pavie A, Derenne JP. Traumatic coronary-pulmonary artery fistula 23 years after a stab wound. Ann Thorac Surg 2000;70:1399–400.
- 112. Kennedy RJ, Clements WDB, Mudd DG. A late complication from a self-inflicted stab wound. Ulster Med J 1999;68:40–2.
- 113. Taupin JM. Testing conflicting scenarios a role for simulation experiments in damage analysis of clothing. J Forensic Sci 1998;43:891–6.
- 114. Scottish Trauma Audit Group. Preliminary analysis of the care of injured patients in five Scottish teaching hospitals: First report from the Scottish Trauma Audit Group (STAG). Health Bull (Edinb) 1995;53:55-65.

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